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ACUTE RHEUMATISM.[1]

By LAURENCE HUGHES,

Honorary Physician, Royal Prince Alfred Hospital; Honorary Physician, Royal Alexandra Hospital for Children; Lecturer in Medical Diseases of Children, University of Sydney, Sydney.

I PRESUME that my remarks this evening are to be confined to acute rheumatism as it occurs in childhood, to which the term juvenile rheumatism, originally introduced by Miller, is now commonly applied. This condition is still an important problem in public health; but in this State it appears to have comparatively little appeal to the public and is only of passing interest to the health authorities. For several years past there have been a few voices crying in the wilderness in this regard, and although something has been achieved, much more could be done with ultimate benefit to child life and adolescence. It has been suggested that I should include modern methods in the treatment of acute rheumatism in this paper. Those of you who may expect to hear of some new therapeutic measure for the treatment of juvenile rheumatism are doomed at the outset to disappointment. We are still largely dependent upon time-honoured remedies in this regard, and any consideration of newer methods of treatment will be along the lines of preventive and public health measures indicated above.

It is my intention to introduce into this contribution as little of science and as much of practice as is possible in the time at my disposal. After brief reference to the ætiology and pathology of acute rheumatism, consideration will be given in more detail to some of the difficulties in diagnosis and treatment which confront those of us who see a number of the victims of this disease in our routine work.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 25, 1940.

Ætiology.

The essential cause of acute rheumatism remains unknown. For many years the streptococcal theory has been the predominant one, and at the present time no one seriously doubts the importance of these organisms in relation to the disease. There are, however, several gaps to be filled in if we are to regard streptococcal infection as the causal agent of acute rheumatism. On the clinical side there is the well-recognized relationship between initial streptococcal throat infections and acute rheumatism in children. But whereas streptococcal throat infections are comparatively common amongst children, the incidence of acute rheumatism as a sequel is comparatively slight. Again, drugs of the sulphonamide group, which are so effective against these organisms in other diseases, appear to be not only of no value in rheumatic fever, but even to exert a harmful influence on patients with this disease. From the bacteriological aspect, the streptococcal hypothesis postulates that rheumatic tissue damage is due directly to microbic invasion; but there is no uniformity of opinion among different observers in regard to the direct demonstration of streptococci in the inflammatory lesions of acute rheumatism. In this regard it is interesting to note that in 17 autopsies on rheumatic fever subjects Collis has recently grown hæmolytic streptococci from the centres of 14 out of 15 tonsils, 13 out of 27 cervical glands, and 22 out of 42

heart valves. Green, on examination of pericardial and valve lesions in nine cases of acute rheumatic endocarditis, has also isolated a hæmolytic streptococcus from the valves in eight cases in which macroscopic valvular lesions were present.

The probability that there are other causal agents in the disease has been responsible for a large amount of modern research, and the immunity reactions to the hæmolytic streptococcus have provided a natural line of investigation in this direction. Schlesinger, Signey and Amies a few years ago showed that streptococcal precipitins could be demonstrated in the blood of rheumatic patients following acute streptococcal throat infections, the most usual time for their appearance being a week or two after the throat infection. This time period corresponds closely to the occurrence of rheumatic relapses after streptococcal tonsillitis. It would thus appear that rheumatic symptoms following a streptococcal throat lesion appear at a time when the immunity response to the same infection is at its height. The authors suggest that "the tissues at that time appear to be particularly vulnerable and this . . . permits an already present but latent causal agent to enter its state of activity". They consider that this agent may be a virus. At a later date the same authors, by means of high speed centrifugalization of pericardial fluid from patients with acute rheumatic pericarditis, obtained deposits, morphologically similar to bodies from known viruses, which on suspension were specifically agglutinated by serum from patients suffering from and resisting an acute rheumatic infection. They considered these bodies to be in the nature of a virus and to represent the actual infective agent of acute rheumatism. This work has recently been extended and carried into the field of chorea and rheumatoid arthritis by Eagles, Evans, Turnbull and Keith. From the evidence thus available it has been suggested that the symptoms of acute rheumatism at the onset of the disease or of its relapses are initiated by a streptococcal infection and continued through its clinical course by the agency of a virus.

Reference must also be made to the suggestion that acute rheumatism is a reaction of hypersensitive tissues to an agent which has specifically sensitized the cells. This is an attractive theory and one which has much to recommend it clinically. But it brings us to that convenient and frequently used term (though not always so readily explained), allergy, which even those who are especially skilled in the study of immunology still regard as a subject of considerable difficulty.

Environmental factors also play their part. Cold and dampness have long been regarded as conditions favouring rheumatic infection, as also has the influence of overcrowding.

Pathology.

Rheumatism affects the mesodermal conective tissues of the body. These are not highly differentiated structures and their reaction to pathological processes is limited. Poynton (1899) first described foci in the interstitial tissue of the myocardium in rheumatic fever, and Aschoff and Tawara (1906) described the presence of nodules in the heart, which they considered the most significant local manifestations of acute rheumatism. Though these structures are most commonly found in the heart, they may occur in synovial membrane and in other connective tissues in the body. Aschoff considered that the primary change was a proliferation of connective tissue cells followed by degeneration and necrosis. Other observers hold that there is primarily a fibrinoid degeneration of the connective tissue ground substance and fibril bundles with exudation into the tissue spaces followed by necrosis. Proliferation of mesenchymal cells and invasion by leucocytes then occur secondarily around the necrotic areas. Subcutaneous rheumatic nodules have essentially the same structure as Aschoff nodules in the heart. In addition to these specific reactions of the tissues, non-specific pathological lesions occur in various tissues, such as the changes seen in the arteries, particularly the coronary arterial system. Exudative, degenerative and infiltrative processes occur similar to those in many general infections. These result in ischæmia of the cardiac musculature, which also undergoes degenerative and atrophic changes as the result of the action of rheumatic toxin.

Blood changes commonly found in acute rheumatism consist of anæmia, mild leucocytosis and an increase in the blood sedimentation rate.

Symptoms and Diagnosis.

The clinical manifestations of juvenile rheumatism are protean in nature, and there is no characteristic onset or clinical course of the disease. There are, however, certain features which are typically associated with one or other of its forms, and these will constitute the main basis for discussion here. The frequent history of a preceding sore throat has already been stressed. This may be followed by an attack of rheumatic arthritis with or without accompanying or subsequent carditis. In other instances carditis may be the only clinical feature of the disease, and its onset may be insidious and only discovered accidentally. Rheumatism may declare its presence by an attack of chorea, with or without carditis. Finally, there is that vaguely indefinite group of subjects, comprising children who suffer from debility and pains in the limbs, to which the term subacute rheumatism is sometimes applied. These varying features of the disease may now be considered *seriatim*.

Arthritis.

It is, of course, well recognized that the joint symptoms of rheumatism in children do not present the frank arthritic manifestations of the disease in adults. The affected joints are always painful, but swelling and redness may be absent. The knee, ankle and wrist joints are probably the most frequently affected. Arthritic involvement may at times be limited to a single joint, which is not infrequently the hip joint. In cases of monarticular arthritis, however, other causes of arthritis should be excluded before the condition is regarded as being due to rheumatism (even though other findings suggestive of the latter condition may be present). This is not always an easy matter, as the following case report indicates.

N.S., a female patient, aged nine years, was in her usual good health until six days before her admission to hospital, when she complained of pain in the left hip joint. On examination she was found to have a temperature of 101° F. and a pulse rate of 110 per minute. Some pain was elicited on movement of the hip joint, but there was no limitation of movement. No other joints were involved. The sedimentation rate was 30 : 50. *Streptococcus hæmolyticus* was isolated from swabbings of the throat. An X-ray film of the hip joint revealed no abnormality. A diagnosis of monarticular rheumatism was made and salicylates were given with a good response after five days. The patient was afebrile for the subsequent four weeks. The sedimentation readings during that time were as follows: 30 : 50, 18 : 33, 8 : 26, 11 : 24, 6 : 12. Before the child was allowed up a further X-ray photograph of the hip was taken; this revealed an active tuberculous lesion.

In the diagnosis of rheumatic arthritis in children the previous history and age period are often of help, as are also the absence of other indications of rheumatic infection and the presence of a much increased sedimentation rate. In monarticular lesions the possibility of osteomyelitis should be excluded; but in this condition the constitutional symptoms and localized tenderness are frequently more severe, and the lesion tends to be more in the region of the bony epiphysis than in the joint itself.

Carditis.

Carditis is the most important clinical feature of acute rheumatism in children, and one which brings it into prominence as a serious disease. It has already been stated that carditis may declare itself as an accompaniment of an acute attack of rheumatism or subsequent relapses, or its onset may be insidious. There is a justifiable tendency of the present day to decry the importance previously attached in clinical medicine to certain cardiac murmurs, particularly those located at the apex. In the rheumatic carditis of children, however, these murmurs are still of considerable interest and

significance. For some time after an attack of rheumatic carditis an apical systolic murmur may be the only evidence of actual cardiac or valvular damage. The apical diastolic murmur, which in adult life is regarded as indicative of organic disease, is not uncommonly a transient murmur in rheumatic carditis and is certainly not necessarily indicative of established or developing mitral stenosis. This feature has been stressed by Sheldon, Schlesinger, Paul White and others, and in my experience it is by no means an uncommon finding. The murmur, which more commonly occurs in the earlier part of cardiac diastole, may disappear during the acute stage of the illness or during convalescence, or it may be heard for several months after the acute attack and then disappear. On the other hand, it may persist indefinitely and later be followed by the murmur which is regarded as being clinically diagnostic of mitral stenosis. Paul White has suggested that this early diastolic murmur may be attributed to the loss of tone and disturbed function of the myocardium incident upon the carditis. Another reason which has been advanced is that it is due to relative insufficiency of the aortic valve and is thus clinicaly analogous to the Austin Flint murmur.

The earliest manifestations of cardiac involvement in acute rheumatism will frequently be heralded by tachycardia. Associated with this there may be clinical evidence of cardiac enlargement with or without a systolic murmur at the apex. At this stage, or subsequently, there is not infrequently heard a third heart sound in early diastole, which may be replaced later by the diastolic murmur mentioned above. These findings may entirely disappear as the clinical condition improves, and there may be no further evidence of the original lesion. In other instances, although the clinical progress is satisfactory in all other respects, one or other of the murmurs or both may persist for an indefinite period. Aortic lesions are not uncommon in children with rheumatic carditis, though they rarely occur during the first attack. They more frequently result from the effect of a relapse on a previously damaged heart. In general, the extension of cardiac disease in children is mostly due to the occurrence of relapses, although mitral stenosis may develop in the wake of a single attack of rheumatic fever. Pericarditis is a severe form of cardiac rheumatism and is often fatal. Rapid enlargement of the heart and liver with increase in the severity of constitutional symptoms are suggestive findings.

Congenital cardiac lesions may at times give rise to difficulty in diagnosis when children over five years of age are seen for the first time with evidence of cardiac

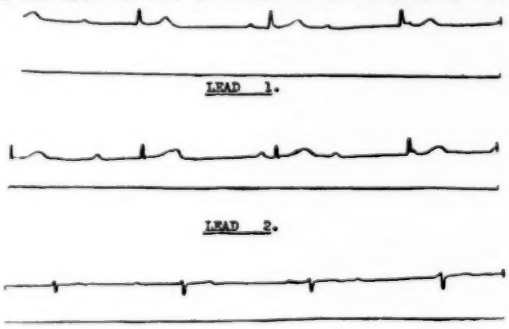

FIGURE I.
Electrocardiographic tracing from a boy, aged ten years, with heart block, who has a pronounced Roger's murmur along the left border of the sternum. (Time markings have been inadvertently omitted.)

enlargement and a murmur, but without other characteristic accompaniments of congenital heart disease. Murmurs of maximum intensity in the intercostal spaces to the left of the sternum, and either systolic or both systolic and diastolic in time, are very suggestive of congenital lesions. The radiological appearances of the heart may help, and there may also be associated characteristic clinical findings, such as heart block in conditions of patent interventricular septum.

Chorea.

Chorea is the most frequent solitary manifestation of all forms of acute rheumatism, and it is now generally regarded as evidence of rheumatic activity in children. Coburn, however, does not consider that it suffices for the recognition of the rheumatic child nor for the diagnosis of rheumatic activity. He points out that of a large number of children under observation for chorea 50% were non-rheumatic subjects, and that the choreic child may have no family history of rheumatism or even show any other signs of the disease. Psychical influences are often important predisposing causes of chorea, and the first symptoms of the condition are not uncommonly detected at school. Among the outstanding clinical features of chorea, mention may be made of its preponderance in girls, of the presence in uncomplicated cases of a normal sedimentation rate during the active stage, of the tendency to frequent relapses, and of the tendency in the great majority of cases to spontaneous cure. This last feature should be borne in mind when, with a view to shortening the course of the disease, consideration is given to special forms of treatment by "Nirvanol" and other methods which in themselves may introduce an element of risk to the patient.

Pains in the Limbs.

Children are subject to pains in the limbs from various causes; but for a number of years these so-called "growing pains" have been regarded as being particularly associated with rheumatism. In some quarters the pendulum has now swung entirely in the other direction, and the pains are attributed in the main to conditions other than rheumatism. No dogmatic statement can be made on the matter. The important thing is to take a careful history and make a thorough examination of every child whose chief complaint is pain in the limbs. When rheumatism is the cause, associated evidence of rheumatic infection is more likely to be present, particularly in regard to blood changes, and the pain is located more in the joints and interferes with their movements, whereas in other conditions the pain is more often confined to the lower limbs and affects the ligaments and muscles. Orthopædic defects (for example, flat-foot and knock-knee) are probably the commonest of other causes of limb pains in children; but they may occur in any condition associated with vague ill health and debility, and in children who are subject to asthma.

Treatment.

The treatment of acute rheumatism consists of the treatment of the acute phase in hospital or in the child's home, organized control of the period of convalescence, preferably in special hospitals for the purpose, and subsequent supervision in a rheumatism clinic. This scheme applies, of course, to those children who for one reason or another cannot be attended throughout the whole course of their illness by their own medical practitioner.

The Acute Phase.

Rest in bed with efficient nursing for a period of six weeks and the administration of salicylates are the essentials in the acute phase. Affected joints should be wrapped in wool and protected from bedclothes by a cradle. The diet should be adequate to meet the child's requirements, with added vitamin if indicated. There seems to be some evidence to suggest that a vitamin *C* deficiency exists in rheumatic fever. The heart should be examined daily for any clinical evidence of involvement in the rheumatic process, and the day and especially the sleeping pulse rate should be carefully recorded. An estimation of the blood sedimentation rate should be made every week.

No other group of drugs has been found to have so much effect on the disease as the salicylates, which were introduced many years ago. They shorten the course of the illness and relieve the associated pain. Sodium salicylate, with an adequate dose of sodium bicarbonate,

should be administered in sufficient amounts to relieve the symptoms without producing any toxic effects. In the case of a child this can generally be effected by doses of 10 to 15 grains every four hours for a few days and then three times a day for a further three weeks. The drug does not appear to have any beneficial action in rheumatic carditis. Aspirin may be substituted for salicylate of soda, in the occasional instances in which the latter is not well tolerated. Sheldon and others have stressed the value of aspirin in the treatment of acute sore throats in children who have had a previous attack of acute rheumatism. Such treatment tends to militate against a relapse of rheumatic fever.

Convalescent Hospitals.

The importance of the proper control of the convalescent period for children recovering from rheumatic carditis was stressed by Poynton thirty years ago, and as a result special hospitals have been established for this purpose in England. These institutions provide a means whereby children can pass though the necessary stage of rest in bed for prolonged periods, followed by carefully adjusted and supervised resumption of activity and at the same time education can be given. Their value is undisputed, both from the point of view of the benefit to children suffering from rheumatism and also because it has been possible through their agency to advance our knowledge of the disease. The main guides to the progress or otherwise of the rheumatic infection are the clinical examination, the recording of the sleeping pulse rate and the weight, and estimation of the blood sedimentation rate. It is important that precautions should be taken against possible reinfection in these homes, and with this end in view sufficient bed-spacing and ventilation are of utmost importance. Ample facilities for the immediate isolation of any child with respiratory infection should be provided, and a close watch should be maintained for carriers of *Streptococcus hæmolyticus* in either the children or staff. The cooperation of the parents may at times be difficult to obtain, as they may not readily agree to their children being away from home for such long periods. The children themselves may find the long restraint irksome.

> And does it not seem hard to you
> When all the sky is clear and blue
> And I should like so much to play
> To have to go to bed by day?

FIGURE II.
Pavilion at the Smith Family Recovery Home.

FIGURE III.
Recent additions to the Smith Family Recovery Home.

This latter difficulty is minimal, however, when all the children are undergoing the same routine treatment. This provides one of the strongest arguments in favour of special convalescent homes for children with rheumatic carditis, who should not associate with comparatively active children convalescing from other ailments. The criticism is sometimes raised that much good done by prolonged institutional treatment is often counteracted when the child returns to an unhealthy home environment; but surely this criticism should be aimed rather at the health authorities than at the convalescent hospitals. Another objection, which may at times be a real one, is that these homes may lay the foundation for a neurosis in the parent or child, or in both. But this should not occur if the physician in charge is awake to his responsibilities. As soon as he is satisfied that the cardiac condition is normal as far as he can reasonably estimate and that no further convalescent treatment is necessary, the sooner the child is allowed to lead a normal life, the better. These objections are, however, all minor ones compared with the immense major benefits which these hospitals provide.

In a previous communication on juvenile rheumatism I drew attention to the establishment in Sydney of the Smith Family Recovery Home for the convalescent treatment of children suffering from acute rheumatism. Useful work is still being done in this institution, and it meets a much-felt need. It is, however, entirely maintained by a charitable organization, and its progress has been considerably retarded because juvenile rheumatism has comparatively little public appeal. There never appears to be any difficulty in raising funds for crippled children; but, strangely enough, the children who are potentially or actually crippled through rheumatic cardiac disease are left to the consideration of a comparatively small number in the community. Fortunately, it has recently been possible to increase the activities of the home by providing accommodation for girls as well as boys. This institution is, however, the only one of its kind in the State, and its present capacity cannot be expected to cope fully with requirements.

Rheumatism Clinics.

The treatment of rheumatic children should not end with treatment during the acute or convalescent stages of the disease. It has already been stressed that juvenile

rheumatism shows a strong tendency to repeated relapses. There is no indication as to the cases in which relapses are likely to occur, as to the time of their occurrence or as to their severity. The relapses, however, frequently give warning signals which can be detected if the child is under supervision. Furthermore, a slowly smouldering carditis may be more readily detected and treated if the child is being regularly examined. For these reasons it is essential for the efficient treatment of children suffering from the disease that prolonged medical supervision should be instituted. This purpose is well served by means of rheumatism clinics or supervisory centres. Clinics of this nature are attached to the Royal Alexandra Hospital for children and to the Rachel Forster Hospital for Women and Children. Apart from the medical supervision thus provided, an essential and very important part of the work in these clinics devolves upon the almoners. It is now generally recognized that social conditions are important factors in this disease, and the efforts of the almoners are directed towards ensuring that the recommendations of the clinic are carried out and adjusting the social conditions, schooling and vocational training problems which may be retarding recovery.

FIFTY-FIVE CASES OF RADICAL OPERATION FOR CANCER OF THE BREAST UNDER LOCAL ANÆSTHESIA, WITH AN ACCOUNT OF THE TECHNIQUE.

By C. E. CORLETTE, M.D., F.R.A.C.S.,
Sydney.

FOR many years I was accustomed to perform all operations under ether anæsthesia. Then I learned that local anæsthesia had great potentialities, and I found that when properly developed and used it made shockless surgery possible. Not only was it safer, but it was more comfortable to the patient, and it abolished much post-operative misery. But local anæsthesia, no less than ether or any other general anæsthetic, or indeed any operation, must be properly administered. It is not to be judged by the amateurish, inadequate, and more or less disappointing procedure that some call and think to be "local anæsthesia". When a man tells us that he has found "local anæsthesia" unsuitable for this or that operation, let us be critical of the critic. We may suspect, with a large amount of probability, that he himself is the failure, not local anæsthesia. Somebody else would have done better.

My first radical breast operation under this technique was on a woman of seventy-nine who refused any operation unless it could be done with a local anæsthetic. The challenge was accepted. The operation was wonderfully successful, and thus encouraged, I tried it on others, at first only on the old and feeble, but again always successfully. In case after case one would find a woman of advanced age leaving the operating theatre apparently as well as when she entered it, with a firm, steady, unaltered pulse, often around 80 per minute. Shockless surgery had come. The contrast with ether was too convincing to withstand. An hour to an hour and a half of ether anæsthesia for a patient around eighty years of age, especially for an operation in which a good deal of blood can be lost, is a very serious matter. Such patients do not come back with a good colour, a good pulse, and apparently as well as when they went into the theatre. They are knocked out.

Moreover, after local anæsthesia the patients were very well, pleased, grateful, and comfortable. I had to ask myself why others should not have it. If it was so good for the old and feeble, it must be good for the others. The question must no longer be: "Can she stand ether?" nor must it be: "What suits my convenience best?" It must be: "What is best for her?" It could not be withheld.

I have not forced it on patients. If reasonably fit for it, they could have ether, and I have told them so. But I have told them that local anæsthesia was safer and more pleasant for them. Once they knew something about it, not one has wanted ether. In the wards, women find satisfied patients who have had other major operations performed in the same way, and they meet nurses and ward sisters who can tell them of many more. It can be said, further, that they are never let down. What they are promised, that they get.

I have now performed 55 of these radical breast operations under local anæsthesia from beginning to end, and I have piloted friends through perhaps half a dozen more. There have been no failures. In no case has it been necessary to call in the aid of a general anæsthetic.

If one surgeon can do this, so can others. That does not mean that an untaught novice, without any other experience in local anæsthesia, is fit at once to begin with the removal of a malignant breast as his first essay in the method. But he can learn. It is worth learning. I suggest that there is material here for serious consideration. It is a technique of shockless surgery, well tested, and successful.

There was at first one great bugbear, and that was the time, trouble, and patience required by the injections. The time occupied for a breast case has often been noted, and it has usually been around thirty-five minutes. It was tedious, though at first it was unavoidable. But when at length a good dependable routine technique seemed to have been reached, the time came to teach, and pupils being eager to learn, there was no more difficulty. From that time onwards the burden of giving the injections passed to an assistant. Definitely, that should be the rule. The operating surgeon should indeed always be competent to do it, but it ought not to be his job. Now the injections are either given by the first assistant or are given by a learner under his tutorship. In this way the resident medical officers soon become quite expert, and of course what applies to removal of the breast applies also to cholecystectomies and other operations.

The injections are obviously a surgical procedure. Moreover, we plan to achieve more than one purpose in the injections, or at least we do so in the technique developed here. The adrenaline component of the solution is wielded as a special surgical instrument, a physiological tourniquet. It is natural and proper for the injections to be the duty of an assistant with surgical training, a surgical outlook, and a sufficiently competent knowledge of the applied surgical anatomy of nerves, arteries, and tissue planes.

While the thirty-five minutes or so taken by the injections is tedious, and much longer than the time taken for the induction of ether anæsthesia, there is a set-off in the lessening of the time taken for the actual operation, and therefore an economy of time to the surgeon when the injections are undertaken by an assistant. That is because of the hæmostatic influence of the injections, at least as employed in the series here concerned. While cases occur that keep one busy with hæmostatic forceps, the tendency to hæmorrhage is not so great as is commonly found in patients under ether anæsthesia. An operation that would take seventy or eighty minutes under ether anæsthesia will take fifteen or twenty minutes less under local anæsthesia. Few take more than an hour, and the majority are well below that. The saving of time is in the removal of breast, muscle, and axillary contents, and not in the suturing. The saving of time to the surgeon is of relatively little importance, but the saving of blood loss is an important gain to the patient.

Premedication.

In these and other operations efficient premedication is a fundamental preliminary to the whole procedure. Furthermore, the method must be suitable to the occasion. Drugs that are liable to cause restlessness may not matter if a general anæsthetic is to follow, but restlessness under local anæsthesia is a serious matter. That fact excludes paraldehyde and the barbituric acid derivatives. Uncontrollable restlessness is too common with them. As regards paraldehyde, I speak with adequate personal experience, but as regards the barbiturates, I depend on reports.

Fortunately, a properly conditioned combination of morphine and hyoscine affords us nearly all we want. It is not perfect, but it is very good. That, however, depends on the conditions observed. The conditions are that the drugs must be used as experience has shown that they ought to be.

I discussed premedication for local anæsthesia at some length in THE MEDICAL JOURNAL OF AUSTRALIA of July 6, 1935. Amongst other things, I discussed the myth of idiosyncrasy as regards hyoscine. The rational explanation, derived from an observed series of over 2,000 cases, is that tolerance follows a regular curve for age, being approximately two and a half times greater at age twenty-five years than at age fifty. A tolerance or intolerance that is orderly, regular, and predictable is not idiosyncrasy. Idiosyncrasy implies the reverse. None of these 2,000 people showed it, and 2,000 is a large number. Here (Table I) is the schedule of dosage recommended for female patients from age thirty years onwards, which is the part of life that matters in the present connexion.

TABLE I.

Schedule of Premedication Dosage for Females Aged Over Thirty Years.

Ages.	Time Given Before Operation.	Dosage in Grains.	
		Morphine.	Hyoscine.
30 to 34	Two hours. One hour.	1/3 1/6	1/100 1/300
35 to 49	Two hours. One hour.	1/3 1/6	1/100 None
50 to 74	Two hours.	1/3	1/150
75 to 84	Two hours.	1/4	1/200

Drugs for Local Analgesia.

For the analgesic drug I have invariably used ethocaine, given in association with a 1 in 200,000 solution of adrenaline. I use always the name "ethocaine" in preference to "procaine", its pharmacopœial synonym, because "procaine" is a dangerous name that ought to be dropped. It has been responsible for many fatal accidents. That is because the name, if indistinctly heard, is easily confused with "cocaine", and cocaine is at least five times more toxic. Moreover, "procaine" and "Percaine" sound deceptively similar, and in handwriting it may be impossible to decide which was meant. "Percaine" is at least as toxic as cocaine. "Percaine" is "nupercaine" in America, and (I think) in South Africa. On the telephone, it may be difficult to distinguish between "nupercaine" and "Novocain". "Novocain", "Planocain", and "Kerocain" are proprietary brands of ethocaine. One brand may cost less or more than another, but that is all.

The solution to be injected should be isotonic. To be isotonic, a 0·5% solution of ethocaine should contain 0·8% of sodium chloride. This can be prepared from normal (0·91%) saline solution by making 100 parts of normal saline solution up to 112 parts by the addition of boiled water. A 0·75% solution of ethocaine is isotonic in 0·75% sodium chloride solution, but 0·8% is near enough. A 2% solution of ethocaine is isotonic when the sodium chloride solution is 0·55%. A 0·55% solution of sodium chloride can be made by diluting 100 parts of normal saline solution to measure 163 parts. We are indebted to R. A. Fulton[1] for the experimental work on isotonia in this connexion.

Neither adrenaline nor ethocaine should be boiled. The 1 in 200,000 solution of adrenaline, as I have found, is soon rendered inert by boiling. By boiling a solution of ethocaine, the pH is lowered materially, indicating a change towards acidity, with a corresponding structural alteration.

We can reasonably assume that sealed bottles of standard adrenaline chloride solution are free from organisms dangerous to wounds. Tablets of ethocaine, or ethocaine and adrenaline, that have been in dry storage for four months are sterile unless they have contained sporing organisms. The same applies to ethocaine powder stored under dry conditions in bulk or in gelatine capsules. Some have feared contamination with tetanus spores from gelatine capsules. This is an abstract possibility, but so improbable that ordinary reasonable men would not consider it. In any case, no danger from sporing pathogenic organisms is likely to be prevented by ordinary boiling. Boiling has an impressive mental effect on the surgeon, but not so much effect on spores. Tablets and powders treated by dry storage are as sterile as a surgeon's newly boiled gloves. If a surgeon requires absolute spore-free sterility for his solutions—a sterility greater than that of his gloves—there is only one way. He must use a Seitz filter.

Surgical Anatomy.

Before coming to the injections it will be profitable to sketch some of the surgical anatomy. Local anæsthesia technique, as I have tried to develop it, has always had a double aim, control of pain first, of hæmorrhage next. This is made possible by utilizing the ethocaine for analgesia, and the associated adrenaline for ischæmia. The double aim has useful applications in the breast operation.

We note, therefore, a nerve supply through derivatives of the brachial plexus to muscles connected with movements of the shoulder, such as the two pectorals, the latissimus, and the subscapularis. The cutaneous nerve supply is derived from the intercostal nerves, excluding the first, but a further cutaneous supply proceeds from descending branches of the cervical plexus, reaching as far as the third rib. With proper care the whole of these nerve paths can be effectively blocked.

Blood supply from behind comes through the intercostal arteries, which give off muscular branches (some of which go to the pectoral muscles), lateral cutaneous, and mammary, the mammary branches being derived from the arteries of the third, fourth, and fifth spaces. Blood supply medially comes from the internal mammary artery, chiefly through its second, third, and fourth perforating branches. These all give branches also to the pectoralis muscle and the skin. From above come, firstly, the pectoral branch of the thoraco-acromial, which runs between the two pectoral muscles, giving supplies to both, secondly, the lateral thoracic, an important vessel giving supplies to axillary contents, pectoral muscles, and lateral part of breast, and then, thirdly, comes the thoraco-dorsal continuation of the subscapular, near the axillary border of the scapula. Some control is possible over most of these vessels, results being better in some than in others, but appreciable always.

Analgesia: Injection Technique.

The technique is designed to meet the indications disclosed by the anatomical survey. The following solutions are prepared: (i) for brachial plexus block, 30 mils of 2% ethocaine in 1 in 200,000 adrenaline and 0·55% sodium chloride solution; (ii) for intercostal nerve blocks, 120 mils of 0·75% ethocaine in 1 in 200,000 adrenaline and 0·8% sodium chloride solution; (iii) for all the rest of the injections, 200 mils, 250 mils, or more (according to the size of the patient) of 0·5% ethocaine in 1 in 200,000 adrenaline and 0·8% sodium chloride solution. These solutions are placed in glass beakers suitably marked for identification.

1. With the patient lying supine, on a well-padded table, the brachial plexus block is carried out. The wheal of entry is a finger's breadth above the middle of the clavicle. The guide to the plexus is the first rib. When this is located by the needle, 10 mils are slowly injected. Then the needle is partly withdrawn, slightly altered in angle, and advanced again to the rib in a new but near-by situation, where a second 10 mils are injected. A third 10 mils are injected in a third near-by situation. If blood should show through the needle, its position should be altered till no blood comes. If seen (and I have never seen it) it would probably be due to a wound of the transverse cervical vein. No injection is ever given with a still needle, but with a short to-and-fro movement. This prevents accidental intravenous injection.

2. A second table is now placed beside the original one, of the same height. The patient is carefully rolled on to it, into a prone or nearly prone position. The arm is placed so as to keep the scapula well away from the mid-line. The prominence of the spinous process of the seventh cervical vertebra is identified. This corresponds with the level of insertion of the needle for blocking in the first intercostal space. From this point a line is marked downwards as far as the eighth rib at a distance three fingers' breadth from the mid-line. A wheal is made with a fine needle either at the top or about half-way. Through this a sufficiently long needle is introduced downwards, or upwards and downwards in the line, and immediately beneath the skin, and 0·5% solution is injected during withdrawal at the rate of one mil per centimetre. This produces an insensitive line in which the needle-pricks for the nerve-blocking injections will not be resented, provided they are kept strictly in the line. Two needles, not too long, are used for the injections, the first not being removed until the second is in place. This is the only certain way of preventing doubling and missing. The first two ribs lie deep, and are the only difficult ones. Since the first intercostal nerve is not concerned with cutaneous sensation, and the artery of the same space is almost equally irrelevant, it is not necessary to deposit any solution in the first space. For all that, it is necessary to locate the first space with the needle in order to give a starting point for the series. Introduce the first needle perpendicularly at the above-mentioned level at the top of the line, and feel for the first rib. When the point touches it, the patient may stir slightly, because periosteum is sensitive. Having found the rib, search gently for the lower margin and place the needle in position with the point just resting against the edge. The needle is left in as a guide, no injection being given. The second needle is now used to find the corresponding position in the second space. The needles are introduced and placed by the right hand while its lower border is resting on the patient's body. When they are in position, the left hand takes hold, supported in the same way, while the right hand takes the syringe. The syringe is gently attached and locked on. The weight is to be supported and controlled without tremor and without jerking. Then the syringe is emptied while the needle is moved to and fro half a centimetre or so, sliding all the time in contact with the bone. The reason for this has been already mentioned. The fluid disperses into the space containing nerves and vessels, blocking pain sense in the nerves, and causing ischæmia in the arteries and veins. The needle in the first space is now withdrawn, while the second remains in place as a guide to the third. In this way the injections are repeated as far as the seventh. In a few cases the eighth space has been injected, but it confers no advantage. In a few cases 0·5% solution has been used instead of the 0·75% strength, and all has gone well, though I still keep to the 0·75%. A syringe graduated to 10 mils holds 12 mils when quite full. A whole 12 mils is injected. It is imperative to succeed in each injection, and anyone who is willing to be careful can make sure of success.

3. When the posterior intercostal injections are finished, the patient is rolled back onto the first table again, into a supine position, for the remaining injections. Any of the 0·75% solution left over is poured into the third beaker. A wheal is made over the clavicle. Through this a sufficiently long needle is inserted towards the shoulder, as far as the acromion. During withdrawal (all such injections are given during withdrawal) a line of 0·5% solution is deposited at the rate of not less than 1·5 mils per centimetre, or more in obese women. In the obese the line should run deep in the fat, because the nerves are in the deepest part. The needle is now introduced in the reverse direction, and a continuation of the line is run to a point on the head of the clavicle of the other side. When this is done, the needle is inserted through the end of the anæsthetized strip near the head of the clavicle, and passed down at a right angle to its former direction. A line of injection is run downwards parallel to the sternum, and about a finger's breadth from it. When it reaches the costal margin the direction is again changed, the line being run across the epigastrium and thence along the course of the eighth rib of the affected side. It continues along the rib as far as a point well over the edge of the latissimus, and there stops. This long encircling line blocks the descending cervical nerves as it crosses their path over the clavicle. Along the eighth rib it blocks overlapping supplies from the eighth and ninth intercostals. In its intervening part, parasternally and across the epigastrium, it produces analgesia sufficient for any ordinary amount of undercutting. As a further effect, we can expect the encircling injection to diminish in some degree hæmorrhage through blood supplied from across the border.

Injections for Restraint of Hæmorrhage.

This completes the analgesic injections. Now follows a series planned to restrain hæmorrhage. These are: (i) internal mammary, (ii) pectoral, (iii) thoraco-dorsal, and (iv) lateral intercostal. They utilize the "physiological tourniquet" effect of adrenaline, and could be carried out just as well with simple 1 in 200,000 adrenaline solution, though personally I have always used the familiar solution of 0·5% ethocaine in 1 in 200,000 adrenaline solution. Since the injections are made in an already anæsthetized field, preliminary skin wheals are not necessary.

The internal mammary injection is intended to influence the second, third, and fourth perforating branches of the internal mammary artery, and the parent trunk itself. The vessel, with its veins, passes down a finger's breadth from the sternum immediately beneath the costal cartilages and the internal intercostal muscles. In the third and fourth interspaces the fibres of the *transversus thoracis* muscle lie behind it. The needle is passed in an oblique direction from below upwards and backwards at the proper distance from the sternum, so that the point shall come to lie behind and in touch with the cartilage. It is advisable to fit the syringe on as soon as the needle-point reaches the depth-level of the cartilages, and to inject slowly as the needle passes into place, where the injection is completed with a to-and-fro movement. The discharge during movement preserves the veins from injury and pushes away the pleura, if too close. Six mils and upwards are injected for each space, making a total of at least 20 mils devoted to this part of the blood supply.

The pectoral injections are carried out as follows. The first, of five mils, is given into the substance of the greater pectoral muscle at the site where it is customary to sever it. For the second, of 10 mils, the needle is advanced beneath the margin of the muscle medially for two or three centimetres, where the injection is given with the usual to-and-fro movement. This injection is designed to disperse fluid into the territory of the pectoral branch of the thoraco-acromial artery. After this, for the third injection, the needle is withdrawn nearly to the surface, and advanced again in a direction still beneath the greater pectoral, but more downwards and more deeply, while avoiding the axilla otherwise. Here 10 mils are injected. The solution is deposited in this situation in order to influence the lateral thoracic artery. After the injections, the parts are gently kneaded to disperse the fluid more widely.

For the thoraco-dorsal injection, also of 10 mils, the needle is inserted beneath the posterior margin of the axilla for a short distance. The solution is discharged slowly into the space between scapula and ribs, where it disperses into the vicinity of the thoraco-dorsal artery.

The lateral intercostal consists of an injection of six or seven mils in the fifth intercostal space, and another, not always given, in the fourth. The third is too high up. The injections should be given from in front of the axillary border of the scapula while the arm is well abducted and elevated. One must avoid passing the needle through doubtful tissue. These injections are designed to augment the ischæmic effect of those already given behind. The needle is inserted and the injection is given with the same technique as at the angle of the rib.

Accessory Technique.

There are no more injections. The surgeon and his assistants now put on their gloves, and the skin is given a final wash with spirit. Provided that certain accessory technique has been attended to, all is ready for the operation to begin.

These accessory details are not to be omitted. They are set down here. (i) The necessity for a well-padded table has already been mentioned. Patients complain greatly if the table is too hard. A spongy rubber top cover satisfies requirements. (ii) A broad band is passed across the thighs and fastened beneath the table. It should not be too tight. (iii) A leather wristlet, lined with soft wash-leather, is buckled round the wrist of the sound side, and then attached to the table by suitable means. (iv) A specially designed horizontal metal shield is attached to the table to guard and keep up the forearm and hand of the sound side. If this is not done, there is a constant tendency for the forearm, wrist, and hand to slip down over the side, there to be pressed on uncomfortably by the body of the assistant. This causes much complaint. Whilst discomfort arising from this cause is most common in abdominal operations, then involving right and left side alike, it is no less to be avoided in an operation on a breast, on the side exposed to it. Patients who have had premedication never complain of the restraint on wrists and thighs. (v) A wire-frame ether mask is placed over the face, and attached to the cheek by adhesive plaster. Over it is spread a gauze drape, and over all a sterile towel. Patients object strongly to the spreading of a drape over the face without an intervening frame. I have not used a transverse screen.

During the operation, an attendant should stand near the head of the patient to watch her and look after her wants. She may ask for her nose to be scratched, or she may feel dry in the mouth from the hyoscine. In that case the nurse lifts up the drape and squeezes some water from a wetted cotton-wool mop onto the patient's tongue. Vomiting during the operation is very rare, but it has occurred. Some mild vomiting after return to bed perhaps occurs in some 25% of cases. But as a rule, the patient is ready for a cup of tea as soon as she gets back to bed.

It should be mentioned that needle-pricks are disliked, and are often remembered, forming the only disagreeable memory of the operation. By taking thought, the number of needle-pricks can be reduced to a very small number, and by skill in producing the initial wheal the discomfort is mitigated.

During the operation the patient lies usually in a state of quiet euphoria. Sometimes she is very sleepy or asleep. Occasionally she is garrulous, querulous, or maudlin. Restlessness is extremely uncommon, and if it occurs it is not seriously troublesome. Now and then there are some flushing and quickening of the pulse, apparently due to the hyoscine.

Summary.

An experience of 55 cases of radical operation for cancer of the breast is recorded, all successfully accomplished under local anæsthesia. In no case was general anæsthesia required. The preliminary injections were undertaken by a surgical assistant, and occupied, on the average, about thirty-five minutes. Not all the injections were for analgesia, certain of them being designed to restrain hæmorrhage through the ischæmic effect of the adrenaline. The patients, many of whom were of advanced age, retained a good colour and pulse, and were never "knocked out". Hæmorrhage was diminished, and this enabled the operation to be shortened by fifteen or twenty minutes, as compared with operations under ether anæsthesia. The operation, as thus carried out, is regarded as an enormous improvement in surgical procedure. The relevant surgical anatomy, the system of premedication, the technique of injection, and the general management of the operation are given in some detail.

Reference.

[1] R. A. Fulton: "Isotonic Solutions for Local Anæsthesia, and their Preparation by Simplified Methods", *New Zealand Medical Journal*, Volume XXX, October, 1931, page 268.

PINK DISEASE: A CONSIDERATION OF THREE ÆTIOLOGICAL POSSIBILITIES.

By F. W. CLEMENTS,
Australian Institute of Anatomy, Canberra.

DURING the last few years three widely different possible causes have been advanced for pink disease: (i) in Jugo-Slavia, Mayerhofer and his associates have incriminated the spores of a cereal smut as the cause of an acrodynia occurring in that country; (ii) a number of isolated therapeutic experiments have led some workers to suggest that vitamin B_1 deficiency is the cause of pink disease; (iii) finally, it has been suggested that pink disease is due to a neurotropic virus.

Smut and Acrodynia.

A disease has appeared in Croatian peasant children which Mayerhofer has associated with the ingestion of cereals (rye) infected with a smut fungus, *Ustilago maidis*.[1] As Australia is an extensive producer of cereals, the question has been raised by a correspondent to the National Health and Medical Research Council whether some cases of pink disease occurring in Australia may be due to the ingestion of smut-infected cereals. It was pointed out that pink disease usually occurred in infants at the age when cereals loomed largest in the dietary, often as prepared breakfast foods made of wheat, maize or oats, not usually boiled.

Dr. Mayerhofer is emphatic that there is only a similarity and not an identity between ustilaginism (as he has termed the Croatian disease) and infantile acrodynia, and affirms that there are patients suffering from acrodynia who have never been infected by grain smut.[1] However, it was decided to repeat with Australian smut-infected cereals the animal experiments which led the Continental workers to state that smut-infected rye was the cause of acrodynia.

In Jugo-Slavia Mayerhofer has incriminated the spores of *Ustilago maidis*, whilst recently in Paris, Debré and Névot[2] have carried out experiments with *Ustilago tritici* and produced toxic results in animals similar to those obtained by Mayerhofer.

The two principal smuts of wheat are the so-called loose-smut fungus, *Ustilago tritici*, and the bunt type, either *Tilletia levis* or *Tilletia tritici*. A crop infected with *Ustilago tritici* is easily distinguished by the black sooty heads containing the spores of the fungus, which, as they are loose and not bound within the pericarp of the berry, are readily scattered by the wind. When dispersed, some spores may become lodged on the flowering parts of adjacent wheat. If the conditions of the wheat are suitable, the spores germinate and a fine mycelium grows down into the developing grain. The fungus then passes into a resting state and the grain completes its development. These infected grains have the appearance of normal wheat, and the fungus does not make its presence known until the flowering time next year. The fungus may be destroyed by soaking the grain in hot water.

Tilletia tritici causes bunt or stinking smut. The fungus grows within the developing wheat berry, and when the grain should be mature, instead of containing the endosperm or "flour", the pericarp contains a mass of black powder, the spores of the fungus. It is conceivable that when such wheat is harvested, the smut balls will be included with the healthy wheat and then pass on to the mills and eventually be mixed with the flour.

There are, then, three ways in which cereals infected with smuts may be eaten: as cereal grains infected with a few threads of a mycelium of loose smut; as loose spores attached to grains; or as smut spores mixed with flour or other cereal foods.

In Jugo-Slavia ustilaginism is caused by cereals (rye) infected with either the mycelia or the spores of the loose smut, *Ustilago maidis*. By virtue of its life history the spores of loose smut would rarely be found as a contaminant of grain. The smut fungi associated with wheat growing are both the loose smut and the bunt type. If either type becomes a contaminant of human food, it would

almost certainly be the spore stage of the bunt type. Despite the life history of loose smut, Mayerhofer, as have Debré and Névot, has used spores of *Ustilago* when testing the toxicity of the fungus for animals. Because of the rare possibility that *Ustilago* spores may affect grain, it was decided in the first instance to investigate the spores of *Tilletia tritici* or *Tilletia levis*.

Mixture of Spores with Cereals.

It had been stated by the plant biologist that because of the objectionable odour of bunt spores (stinking smut) and its colour (black), even low concentrations in flour or breakfast cereals would be easily discernible. To check this, spores from infected wheat berries were mixed with flour. A 1% concentration was obtained by mixing the spores from one infected berry with the flour that would be obtained from 100 berries—that is, with a weight of flour equivalent to 70% of the weight of wheat. Various percentages of infected flour were produced. In concentrations below 1% the spores could not be visually detected. A concentration of 1% produced a slight change in colour to pale grey. The characteristic odour of smut could not be detected under an 8% concentration. These tests were carried out at the end of 1939 with a 1937 crop. A 1939-1940 crop, when tested immediately after harvesting, produced the characteristic odour in a 5% concentration.

It was thought from this simple test that concentrations of about 1% would give the flour a dirty appearance and would lead to its rejection by housewives. Similar results were obtained when the smut was mixed with finely ground breakfast foods.

Toxicity of Tilletia Tritici for Laboratory Animals.

Feeding Experiments.—Debré and Névot used young mice; when the mice were fed on groats containing 8% of smut spores, death occurred in 21 days. This mixture was non-toxic to adult mice and rats. These experiments have been repeated at Canberra, 1937 and 1939-1940 smut balls being used. In addition to groats as the basal diet, mice were also fed on a balanced basal diet containing the following elements:

Yellow maize meal	37·0
Gluten meal	10·0
Casein	5·0
Dried yeast	2·5
Dried milk	10·0
Salt	0·6
Cod liver oil	1·0

To this the smut spores were added. The following results were obtained: (i) The feeding of 8% of spores in groats and in a balanced basal diet was without effect upon adult mice and rats. The health of the laboratory animals differed in no way from the health of the controls. (ii) When the mixture of spores and basal diet was fed to young mice (just weaned) no change was produced. After a month on this diet the weight of the laboratory animals was greater than the weight of the controls. At the end of the experiment all laboratory animals were vigorous and healthy, whilst two of the controls had died from intercurrent infections. From this it may be deduced that an 8% concentration of spores of *Tilletia tritici* was non-toxic for young mice.

TABLE I.
Average Weight of the Animals in One Experiment.

Weeks.	Basal Diet and 8% Smut.	Basal Diet Only.
At commencement	10·7	10·9
First week	12·8	12·9
Second week	14·9	12·4
Third week	14·9	12·9
Fourth week	15·2	13·2

Injection Experiments.—Debré and Névot injected an aqueous extract of spores into both young and adult mice. In the protocols of these experiments the concentrations were not given. Hunt and Thompson[(4)] have recently investigated the pharmacology of *Ustilago*. In their work they used an aqueous suspension of a hydro-alcoholic fluid extract. For these experiments I decided to obtain both an aqueous extract and an aqueous suspension of a 50% alcoholic extract. It was the experience of Hunt and Thompson that these extractions must be made in the dark at low temperature, for if they are carried out at room temperature in the light, toxic elements develop in the extract. I had similar experiences, especially in the preparation of the aqueous suspension of the alcoholic extract. A batch was prepared at room temperature in the light, and about twenty-four hours were required to extract all the alcohol by means of a current of air drawn over the fluid. This batch proved very toxic when injected into both adult and young mice. Further batches were prepared in absolute darkness in an ice box, and injections were carried out as soon as possible after the extraction was completed. Solutions (10%) of aqueous extract and of aqueous suspensions of hydro-alcoholic extracts were used. Intraperitoneal injections of amounts ranging from 0·25 cubic centimetre to 1·0 cubic centimetre were made into adult and young mice without effect. Even when the injections were repeated at daily intervals for a week the solutions were found to be non-toxic. A 25% solution of aqueous extract, which was so syrupy that it just passed through the hypodermic needle used, was without effect when injected intraperitoneally. It was quite evident from these experiments that the toxic effects produced by Debré and Névot could not be reproduced with the spores of *Tilletia*.

Effect on Pregnant Mice.—A mixture of groats to which 8% of spores was added was fed to pregnant mice; it did not produce abortion or affect the vitality and subsequent health of the young mice.

Discussion.

These experiments completely failed to demonstrate the toxicity for laboratory animals of the spores of the wheat smut *Tilletia tritici* or *Tilletia levis* from either 1937 or 1939-1940 crops. Apart from the results of the experimental work, certain other aspects must be taken into consideration when this problem is discussed.

It is the opinion of agricultural biologists that the amount of smut-infected wheat grown in Australia is small and that the percentage of infected flour, if there is any at all, must be very low. Cereal production in Australia is on a very high level; almost all the wheat is sold on the open market, and none is retained by the farmer for his own domestic use; and certainly none is ground up for use in the home as flour.

In Croatia the standard of living is in general very low; almost all the peasants live on the products of their own farms. Most references in literature to the standard of living in Croatia mention the fact that the peasants have very little money and can obtain extra products only by barter. The standard of agricultural inspection is very low, and little is done to prevent the raising of smut-infected cereals or the milling of such grain.

Throughout this experimental work the spores of *Tilletia tritici* have been used. Mayerhofer and Debré and Névot, however, used the spores of *Ustilago tritici* for their animal tests. It is very difficult to understand how the spores of loose smut can contaminate ripe grain in sufficient quantities for it to be toxic when milled and eaten. The only conclusion to be drawn is that the infestation of the crops is so high that when the spores are liberated early in the season they are so dense that they cling in large quantities to ripening grains and so contaminate them.

Summary.

That smut-infected cereals may act as the ætiological factor of acrodynia in Jugo-Slavia has been considered and accepted as a possibility; however, there is no evidence to suggest that smut-infected cereals play any part in the ætiology of pink disease in Australia.

Vitamin B_1 and Pink Disease.

During the last two or three years a number of workers, including Durand, Spickard and Burgess,[(5)] Forsyth,[(6)] Williams, Shapiro and Bartelot,[(7)] and Gretton-Watson,[(8)]

have used vitamin B_1 therapeutically in cases of pink disease. Up to the present time a report has not been made of a series wherein only alternate patients have been so treated.

Forsyth has now placed on record six cases in which amounts of vitamin B_1 varying from 180 to 1,500 international units per day were administered. The results varied; but Forsyth gathered the impression that the condition of the infants receiving the larger doses was definitely improved by this form of therapy.

The two patients reported by Durand, Spickard and Burgess showed improvement when given 2,000 international units a day by subcutaneous injection; but this improvement ceased when oral doses of 600 international units a day were given instead of the injections.

Gretton-Watson failed to notice any improvement in one child who was given 750 international units of vitamin B_1 per day for a week. However, this child's condition improved during a second course of treatment with the vitamin when 500 international units a day were given.

Williams, Shapiro and Bartelot administered two milligrammes (666 international units) of "Betaxan" twice a week by intramuscular injection to a child during the third month of treatment for pink disease. This treatment was followed by an almost immediate improvement in the patient's condition, which was maintained.

The patients with pink disease so far treated with vitamin B_1 have not demonstrated that the disease is due to deficiency of the vitamin. In all cases the administration of vitamin B_1 was only one of the therapeutic measures used; the others included vitamins *A* and *D* as well as specific drugs, such as digitalis in one case and desoxycorticosterone in another.

The patients cited above who were either given large doses by mouth or were treated by injection showed improvement beyond that expected. This evidence alone is not enough to justify the claim that the disease is due to a vitamin deficiency. A possible explanation for the improvement noticed will be discussed later.

The principal evidence against the avitaminosis theory is that many of the infants who contract pink disease appear to be well nourished and have followed what is considered to be a normal weight curve before the onset of the illness. Opposed to this is the not infrequent disorder of infants who are almost certainly suffering from the effects of suboptimal amounts of vitamin B_1 in their diets and who do not contract pink disease. During the last two years a number of children who belonged to this class have been seen at the infant welfare centres at Canberra. It could not be held that these children were suffering from mild pink disease, since they had none of the classical features of the disease and responded very much more quickly to the specific therapy than is the case in pink disease. This condition is under full investigation with chemical controls, and will be the subject of a later communication.

A Possible Explanation of the Improvement Produced with Vitamin B_1 in Cases of Pink Disease.

During the last few years extensive advances have been made in our knowledge of the part played by vitamin B_1 in the body. With this knowledge it is possible to advance an explanation for the improvement noticed in the condition of patients suffering from pink disease treated with vitamin B_1. McCormick[9] thinks that vitamin B_1 deficiency is an ætiological factor in poliomyelitis and has achieved some notable improvements and even cures in cases in which large doses of the vitamin were given. Similarly, it is possible that a deficiency of vitamin B_1 may be a factor in aggravating the signs and symptoms of pink disease. Some years ago Feer[10] advanced the hypothesis that pink disease affects the vegetative nervous system, whilst recently it has been claimed by Mayerhofer that the lesion is located in the mid-brain. Brock[11] states that pink disease is believed to be due to a neurotropic virus, as poliomyelitis is believed to be.

Peters and his co-workers[12] have shown that vitamin B_1 is a catalyst which completes the oxidation of glucose and that in vitamin B_1 deficiency pyruvic acid accumulates within the blood and the cells of the nervous system. Weiss and Wilkins[13] have affirmed that vitamin B_1 requirements are increased in febrile conditions and in all states of heightened metabolism. A virus disease or any febrile condition (pink disease is accompanied by rises in temperature) would increase the body's requirements for the vitamin, which may not be available when the patient has a suboptimal intake.

One of the characteristic features of pink disease is loss of appetite, which would rapidly aid in the depletion of the body's reserve of vitamin B_1. In these circumstances pyruvic acid and similar substances would tend to accumulate in the cells of the body, further interfering with metabolism. Administration of vitamin B_1 in these cases, especially by injection, would bring about removal of these toxic substances and hence relieve the symptoms and signs not due to the invading organism *per se*. Thus vitamin B_1 would have a definite place in the treatment of pink disease.

The Virus Theory of Pink Disease.

As has been stated above, it has been held by some workers that pink disease is due to a neurotropic virus. There is little evidence to support this belief, except that the disease is apparently mildly contagious and occurs in definite epidemics. Dods[14] has recorded a number of instances in which two members from one family have contracted the disease at about the same time. In relation to this theory the epidemiological features of the two cases that have occurred recently in Canberra are worthy of comment. Canberra is a comparatively isolated city, and it is possible to trace the movements of a young baby. Both children referred to had visited Melbourne just before the onset of the disease. In one case an interval of about three weeks elapsed between the return to Canberra and the insidious onset of the disease. In the other case the child was away for six weeks, during which time it attended regularly at the out-patient department of a large metropolitan hospital for a minor surgical condition. The onset of the illness occurred during the fifth week of the child's stay in Melbourne. In both instances the children were well nourished and healthy before the onset of the disease. The complete absence of other cases in Canberra and the fact that both children had visited a large city and had mingled with other children suggest that the disease was contracted there.

It is not possible to draw any conclusions from these cases, which merely offer some support for the theory of an infective organism as the cause of pink disease.

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INQUESTS AND EXPERIENCES IN THE CORONER'S COURT.[1]

By J. B. CLELAND, M.D.,
Marks Professor of Pathology, University of Adelaide, Adelaide.

THE coroner or crowner and his court have always interested me from my student days. There is a mediæval ring about them, as indeed there is in the law courts in general. The coroner is independent of the law courts, can make inquiries in any reasonable direction he may deem fit, is to some extent untrammelled by the restrictions as to evidence laid down elsewhere, and is in fact a law unto himself. And this is not surprising, for in the beginning he it was who, on behalf of Our Sovereign Lord the King, had to inquire as to the real cause of any death by violence, for the personal chattel from which such death resulted was deodand[2] and forfeit to the Crown. On behalf of the Crown he sought out the cause, and at times doubtless valuable acquisitions—chargers and battle steeds, weapons, and so on—were made by the king. One can quite understand how inquiries into treasure trove (gold or silver in plate or bullion, buried by their original owner, but not articles discarded, stolen or lost) were added to his duties. I do not know why arson came in.

My first experiences were when I was only twenty-one years of age, at the Prince Alfred Hospital in Sydney. Like house surgeons of today, I had to attend the coroner's court not infrequently. At that time a rather interesting series of fatalities was occurring. The old steam trams of Sydney, with an engine in front, were being replaced by electric trams. The front and back of the latter were similar, and as people had for long been accustomed to see a real engine in the foremost position in a moving tram, a number failed to realize that the new electric trams were approaching and not receding, and so were run over. I seem to remember a fatal accident a week and a non-fatal one daily.

As a young graduate of two years' standing, the second resident pathologist of that hospital, I carried out there in the post-mortem theatre my first hundred autopsies. The coroner in those days was a layman, and usually held his inquiries in a special room at the morgue belonging to the hospital, empanelling a small jury of four. These jurymen were such as could be gathered together in the vicinity of the hospital, which was in those days definitely in the suburbs. There were usually several regular *habitués* of the neighbourhood of the Prince Alfred Hotel, opposite the hospital, who kept an eye open for these coronial inquiries, on which to some extent they subsisted. In fact, they almost considered themselves as part of the regular establishment of the hospital, and felt that its honour was to some extent placed in their keeping. The verdicts returned were in accordance with this pleasant fiction, and one would gather from the attitude of the jurymen and from their finding after a death under an anæsthetic that the anæsthetic had rather tended to keep the patient alive than to hurry him to another world.

The coroner's room, which was in front of the theatre where the body lay to be viewed by the jurymen, was a very dark room, used at times for lectures to nurses. A stuffed lay figure was made use of in bandaging lessons. On one occasion a small jury of strangers had viewed the body, an unpleasant and nerve-disturbing business. They tripped upstairs to the coroner's room, more or less upset and "nervy". Unfortunately the lay figure had fallen down, and one of them, groping his way to his place, inadvertently trod on the arm, realized what he thought it was, uttered a yell, and with his fellows rushed to the door, where they all jammed.

Post-Mortem Examinations for the Coroner.

It is by no means always an easy matter to ascertain the most likely cause of death, still less the certain cause of death, even when a post-mortem examination is made with the utmost thoroughness. In the insane this is especially the case. I have a heading "No obvious cause for death, or death obscure", under which have to be placed quite a sprinkling of cases. On the other hand, in the insane I have at times found several different lesions, each capable of causing death and appreciable illness before death; yet the death had been comparatively unexpected.

In every post-mortem examination, whether for the coroner or not, a careful external examination is necessary; one should note the colour of the eyes and hair, the state of the teeth, the apparent age, the presence of wounds or bruises, of scars, birthmarks and moles, and the many minutiæ which suggest themselves if the body is that of an unknown person or is likely to be the subject of an inquest. The observations made must be accurate and as full as circumstances warrant. Hence the necessity of having available an amanuensis, acquainted with medical terms, who takes down the notes from dictation as the examination proceeds; measurements and the exact location of lesions are apt to be forgotten if written up after the autopsy. In all my post-mortem examinations I insist on notes being made at the time. When the examination has been completed, it will be necessary for the pathologist to read over what has been written and to sign it at the time as correct.

Another most important factor in ascertaining the cause of death is that the autopsy shall be performed as soon after death as possible. In cases of criminal abortion this is particularly necessary. We have had a long list of deaths from jaundice due to gas-forming organisms introduced into the uterus during the procuring of an abortion, and the post-mortem examinations in these cases should be made if possible within an hour of death. I do not mean to say that conclusive evidence cannot be obtained from autopsies held even many hours after death. It often can; but the changes produced by decomposition occur so quickly that very important, sometimes all-important, information may be lost through the delay.

It is important not to be hurried when a post-mortem examination is being made. This applies particularly to those made for the coroner. Ample time must be allowed, and one must be prepared for unexpected eventualities which may prolong the examination to two or even three hours. Consequently it is unwise to start work with an empty stomach or late in the afternoon of a strenuous day; however, one should remember how important it is to have as little delay as possible, especially in warm weather.

The autopsy should be carried out whenever possible in a properly equipped post-mortem theatre, with all its facilities, including an efficient attendant. In the country this is, of course, often impossible.

With a relative with whom I was staying I have made such an examination with the body on a shutter in the barn of an inn on the old pilgrim road that led from

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[1] Read at a meeting of the Medico-Legal Society of South Australia on July 5, 1939, at Adelaide.

[2] "By Deodand is understood whatever personal chattel occasions the death of a man without the default of another, and also the instrument with which murder is committed. In England deodands are forfeited to the king, to be applied to pious uses and distributed in alms by his 'high almoner'." (*The Penny Cyclopædia*, 1837.)

I cannot find any reference for the opinion that the ancient office of coroner arose in the way suggested. Most law books under the heading of "coroner" do not mention it, and it is only by inference under "deodand":

"Deodand (*Deo dandum*, Latin). A thing given or forfeited to God for the pacifying his wrath, in case of any misfortune, by which any Christian comes to a violent end, without the fault of any reasonable creature; as, if a horse should strike his keeper, and so kill him; if a man, in driving a cart, and endeavouring to rectify something about it, should fall so as the cart-wheels, by running over him, should press him to death; if one should be felling a tree, and giving warning to company by, when the tree was near falling, to look to themselves, and any of them should nevertheless be slain by the fall of the tree; in these cases the horse, the cart-wheel, cart and horses, and the tree, are to be given to God; that is, sold and distributed to the poor, for an expiation of this dreadful count, though occasioned by unreasonable, senseless and dead creatures: and though this be given to God, yet it is forfeited to the king by law, as executor in this case, to see the price of these distributed to the poor. Cowel."—Samuel Johnson, "A Dictionary of the English Language".

The law respecting deodand ceased to operate early in Queen Victoria's reign.

Dover to Canterbury. There was no door, and one looked out over the undulating country of that part of Kent. A passing traveller had called in for a glass of beer, and as he raised the tumbler to his lips a gush of blood rushed forth and the man fell down dead. We found that an aneurysm of the aorta had burst into the left bronchus and flooded his lungs.

A unique specimen that we have in our museum is from an unfortunate man, too polite to vomit in public. He had partaken perhaps too freely of beer and his stomach revolted. Rushing outside, he constricted his pharyngeal muscles by force of will; but the sphincter at the lower end of the œsophagus relaxed, and the powerful peristaltic waves of the stomach propelled its contents with destructive force into the gullet closed above, producing a fatal longitudinal rent in it.

To return to the conduct of the autopsy. Laboratory facilities for bacteriological, chemical and other examinations should be readily available and at the service of the pathologist. The bacteriologist should, whenever possible, himself take the necessary material for cultures, and in cases of poisoning I have found it on occasions a very distinct help for the chemist from the government department of chemistry to come to the post-mortem room and secure such material as he may deem desirable; at the same time I have discussed the case with him. Books of reference should be consulted if necessary, and so library facilities should not be far away.

Quite apart from post-mortem examinations and the clinical examinations that may have been made in coroner's cases, medical witnesses may be called upon to examine various exhibits for blood stains, in order to give opinions as to whether the exhibits could have been responsible for various wounds, and for other purposes.

I have had to examine a human hair caught in the wood of a blood-stained tomahawk. Was it the hair of the murdered white woman or of the owner of the hatchet, a Chinaman? Mongolian hair is blackish and rounded on cross-section, and so the long hair hangs straight down. Caucasian hair is usually lighter and elliptical on cross-section, and so more or less wavy. The hair was Caucasian and corresponded in colour with that of the dead woman.

In another case, a murder on an outback station in the north of New South Wales, blood and hair were found under an outhouse which was raised from the ground on supports. The blood did not give the reactions for human blood, and the hairs corresponded with wallaby hairs; the dogs had taken a wallaby under the room and consumed it there.

Fortunately, an understanding exists with the coroner so that on communication with the coroner's constable, when a death occurs at the Adelaide Hospital in circumstances which are not quite clear, and when the case may possibly come under his jurisdiction, the necessary autopsy can be carried out practically straight away. It would be a still better arrangement if permission could be given to the medical superintendent or his representative at the Adelaide Hospital and other similar institutions to carry out these necessary inquiries with the speed that is requisite if, in all cases, the real cause of death is to be ascertained beyond all cavil. Such permission, I am sure, would not be abused and would help in the elucidation of crime. It can be readily seen that the coroner could not give official authority in general terms for this to be done; but I presume it could be made known that his authority might be anticipated and that it would later be forthcoming.

I have already indicated the difficulties that often exist in the ascertainment of the real cause of death and the contributory factors. It is a very great help to have present another pathologist of standing or some experienced practitioner with whom the ins and outs of the case under investigation can be discussed. A pathologist working alone, however experienced, may neglect to make some observation which may turn out to be of very great consequence in a post-mortem examination. There is so much that may have to be done that this may readily occur. An examination carried to the minutest detail may run into many hours and may become very exhausting, especially if highly important issues are at stake. The presence or assistance of some other experienced person is therefore of real value, and the extra expense involved is trifling compared with some of the issues involved, especially in the case of murder. It is particularly important that such assistance should be forthcoming in cases in the country. At the mental hospital all deaths are by custom reported to the coroner, though there seems to be no legal requirement to do so, as is the case under the English lunacy acts. The coroner may presumably order a post-mortem examination on all these bodies if he thinks fit; but as a matter of practice the question of holding a post-mortem examination is left to the discretion of the medical superintendent. Patients who die there obviously from natural causes do not necessarily, therefore, undergo a post-mortem examination. In sudden death and in cases of an obscure nature an autopsy is always performed. The post-mortem examinations at the mental hospital and allied institutions are carried out chiefly by myself as professor of pathology at the University of Adelaide. The object in view in having these post-mortem examinations done by someone who is not on the staff of the mental hospital or, as a matter of fact, under direct government control, is to satisfy the public that all care is exercised in the treatment of these unfortunate people. If ill treatment existed it would tend to be revealed in the course of systematic post-mortem examinations. It is also necessary to see that the patients in this institution are protected against each other, as homicidal tendencies are not uncommon in some of those thus afflicted. During the course of the post-mortem examination notes are, of course, made of bruises or any other injuries that may have been due to violence; these are all recorded, and a copy of the autopsy report is forwarded to the coroner with my interpretation of the cause of death following on the summary of the lesions found. Bruises and other injuries are by no means uncommon, and the explanation in nearly all cases is self-evident in the nature of the mental illness. Maniacal patients may injure themselves, and bruises are consequently common. In spite of the most watchful care, attempts at suicide and homicidal acts are inevitable from time to time. There is a golden rule for those who have anything to do with the insane—and the same applies to Australian aborigines, even though they may seem more or less civilized: never let such a person go behind you or walk behind you. The homicidal impulse may be overpowering. I remember many years ago the death of a pastoralist in Western Australia who was killed by a black boy who had been in his employ for many years. The impulse to slay, given an especially favourable opportunity, cannot sometimes be resisted.

Only a few months ago a small, comical-looking, old aboriginal named Quartpot, with hardly any knowledge of English, was tried with two or three other natives for having killed another who was believed to have "boned" a relative of one of the party. Quartpot had rather an evil reputation in the district from which he came. He was an expert spear thrower and there were good grounds for thinking that various various native women and children who had mysteriously disappeared had fallen victims to his expertness.

It would appear that, given a favourable opportunity and target, and the spear in his hand, the impulse was irresistible and the spear was launched with the inevitably fatal result. Primitive man and the insane have much in common.

Surprises occur from time to time during post-mortem examinations, more particularly so at the mental hospital, and one must be prepared for them in all autopsies.

On one occasion a man was found dead during the course of the night rounds in a room occupied by several other insane patients. As he was an elderly man, there was no suspicion of anything other than natural causes for his death. Many people with coronary disease die peacefully in their beds. At the autopsy, as usual, a note was made of the external appearances. I failed to detect a very minute puncture through the centre of the sternum. The thoracic cavity was opened, and when the sternum was reflected a little red spot was noticed, but thought to be of no significance. When the pericardium was opened a large quantity of blood was found in this space; death was due to this hæmorrhage interfering with the action of the heart. There was also some blood in the portions of the lung overlapping the heart. The bleeding was traced to a little slit or tear near the commencement of the aorta. There was

no disease condition associated with this tear, and inspection now revealed a punctured wound passing through the skin and the bone of the sternum and puncturing the pericardial cavity to pierce the aorta. In seeking round for a possible implement that could produce such a wound, no weapon of any kind having been found near the dead patient, I had to consider what tool might conceivably have been available. Long mattress needles had been in use in one of the wards, and the puncture looked as if such an implement might fit it. A mattress needle was obtained and exactly fitted the hole right through the bone.

Whether such a weapon was actually responsible for the death we do not know. Every possible place was searched for the weapon that had been used; nothing was found in the room, although, of course, inasmuch as the patients had dispersed in the ordinary way long before any suspicion of foul play had arisen, such a weapon could easily have been removed and thrown away. Even the ventilators near the ceiling were searched to see whether anything had been placed in them. No needle was apparently missing, no other implement could be found, and the only evidence that a mattress needle had been used was the way in which the needle fitted the aperture. Moreover, in this case we did not know whether the patient had stabbed himself or had been stabbed by another patient. That may seem surprising; but the position of the wound in the aorta was such that it is possible that the patient may have been stabbed hours before he died. The little slit had not remained open, so that no leak occurred, or there was only a very slight escape of blood. In the recumbent position, when he lay down at night, it is possible that the orifice may have relaxed so that the pericardial cavity filled with blood. This would explain how it was that an opportunity was given to dispose of the weapon, whether the patient committed the act himself or whether someone else was responsible. A quick stab may have been made during the day-time, with immediate withdrawal. It is possible that if the wound was made by someone else the victim hardly realized that he had been stabbed, so rapid may have been the blow and the withdrawal. It must, I think, have inevitably been a very rapid stab. This is shown by the fact that a thick layer of bone was penetrated. If the mattress needle was used, it seems remarkable, unless it had been hafted to a handle, that it could have penetrated the bone without slipping through the hands of its user. The case altogether was a mysterious one and was never solved.

At the Adelaide Hospital surprises are, of course, continually met with, and of the bodies that come to autopsy there are only about 70% that have had a reasonably correct diagnosis made during life. This is owing to the fact that persons come to hospital quite frequently in the article of death or die within twenty-four hours in circumstances preventing a full and complete examination. There are always many cases of an obscure nature. A diagnosis has to be made from the outside; we cannot handle and inspect the internal organs, except occasionally surgically, during the life of the patient. When one considers the difficulties, it is remarkable that so many accurate diagnoses are made. Cases of suspected poisoning or of possible poisoning arise from time to time, and sometimes poisoning cases are quite unsuspected. There was one in which "Lysol" or arsenic had been used, and the nature of the illness was realized only when the stomach was opened.

I remember Dr. A. A. Palmer, of Sydney, whose experience of coroners' cases is unrivalled perhaps in Australia, telling me how important it was never to infer, from the circumstances in which the body was found, that death was due to these circumstances. He instanced the case of a body recovered from the sea at Manly or one of the other seaside resorts of Sydney. Naturally the inference was that death had occurred from drowning, probably accidentally. The post-mortem examination, however, revealed a revolver wound in the roof of the mouth, entering the brain. The man had waded into the sea and fired the revolver into his open mouth, in the hope that the suicidal act would escape notice and that death would be attributed to accidental drowning.

It is therefore important, however seemingly obvious is the cause of death, that every detail of a full examination should be carried out in all cases.

From time to time difficulties arise in interpretation, however; some explanation of death may be found which seems to have no connexion at all with, say, a previous act of violence, or the nature of the association is obscure. Here all one can do is to present the facts, and then one's interpretation of these facts, keeping clearly distinct the one from the other, as the onus then lies on the unfortunate coroner to give a decision if the position after the interpretation is still obscure. One realizes how difficult it must be for the coroner, unless he is a medical man of wide experience, to assess the value of medical evidence presented to him, especially if there is a diversity of opinion. In all difficult cases, particularly those of possible murder, I am sure it would be a great help to the coroner and would further the interests of justice if there were associated with him a specially qualified medical referee who would assess the evidence and advise the coroner as to the relative values. It would be a moot point as to whether such assessment should occur in open court or whether the coroner, in coming to his conclusion, would discuss the pros and cons *in camera* with the medical referee. Such, I think, would be the proper course to pursue, the medical referee being, in fact, an extension of the coroner.

The medical witness in all coronial inquiries must divest himself of all possible bias. For Crown witnesses this is usually relatively easy to do, as the Crown has no object in view beyond that of ascertaining the truth. In the case of a medical witness whose opinions are sought really on behalf of some possibly incriminated persons, the same attitude must be adopted. Nothing can, in the end, possibly be gained by drawing red herrings across the track; this is quite apart from the honour of the profession, which insists on the giving of perfectly candid evidence. Nevertheless it is at times difficult not to be carried away. This is especially so if there is harassing cross-questioning, which is apt to give rise to a combative spirit in the medical witness. Another thing to be deplored is the making of too emphatic assertions; one looks at dogmatic statements with a great deal of hesitancy.

Another interesting aspect of coronial work is what constitutes a death "not from natural causes". A charge of murder cannot lie if the victim dies after a year and a day have passed. After that period, if the person dies clearly as a final result of some injury received, should the coroner hold an inquiry?

A few years ago a remarkable example of a yeast infection (torulosis) of the brain occurred in Adelaide. The yeast cells, surrounded by large mucinous capsules, were growing into the brain tissue, displacing the nervous structures and "white-anting" the infected areas so that little cavities could just be seen with the naked eye. When the man eventually died a mucinous yeast-infected mass was found in the middle lobe of the right lung and a revolver bullet in the lower lobe below it. It is quite likely that the revolver bullet first introduced the yeast cells into this man's tissues. When had it been fired and had it been fired in malice? On inquiry we found that it had entered accidentally and many years earlier.

Strictly speaking, did this case come under the jurisdiction of the coroner? Was it really a death from natural causes?

Tetanus is often due to a trifling wound or prick, or no obvious point of entrance of the organism may be found. The focus of entry may sometimes be in association with the teeth. Deaths so caused I should call deaths from natural causes, as distinct from deaths due to tetanus developing as a result of criminal abortion or after a road accident.

At one time it was a requirement in New South Wales that all deaths under an anæsthetic should be reported to the coroner and an inquest should be held. I doubt whether this was a wise procedure. No medical man likes an inquest in which his actions, however justified, may be subjected to criticism in a tribunal open to the public. In desperate cases, which are not uncommon, an operation may be the only possible means of saving life. Death may result on the table and under the anæsthetic, and the death may be entirely due to the cause requiring the operation or may be precipitated by the anæsthetic.

If an inquest is likely to be held should a death occur in these circumstances, the surgeon may be inclined to hold his hand (death being seemingly inevitable), and a last possible chance of saving life may be lost. Or, death being imminent, he may hurriedly "sew up", leaving his work uncompleted, in the hope that the patient may leave the operating theatre alive and so far return to consciousness as not to be under the anæsthetic. Such possibilities, which raise subsidiary issues at a time when all the mental faculties should be focused in doing what is best for the patient, are to be deplored.

Conclusion.

Finally, let me make a suggestion which is really barely germane to the subject before us. Motor car accidents come our way, be we lawyers or medical men. We all run the risk of collisions. May I suggest that these questions should be put to drivers in the latter circumstances: "Did you do the right thing yourself?" "Were you prepared for the other person's doing the wrong thing?" Times advance and circumstances change. To err is human. In the words of Hippocrates: "Experience is fallacious and judgement difficult." One has frequently seen at intersections reckless motorists relying on their right of way, jeopardizing human lives. May I suggest that the old legal dictum that everyone is supposed to know the law and abide by it, or take the consequences, should not be allowed any longer as an excuse in such circumstances, and that the failure to be prepared for error on the part of the other driver should be in itself culpable.

In conclusion, I should like to pay a tribute to the present administration of the coroner's office. There is complete confidence between the medical profession and the coroner in South Australia. This confidence is such that on one occasion, in an important and very delicate matter, a colleague and myself had no hesitation in journeying a considerable distance to visit the coroner when he was on holiday, to place the new position that had arisen before him and to ask his advice as to how we should act.

GAS TENSIONS IN THE TISSUES OF NORMAL RESTING SUBJECTS.

By ERIC W. SIBREE,
From the Department of Medicine, University of Sydney.[1]

CAMPBELL has given, in *Physiological Reviews*,[(6)] a comprehensive review, up to the date of publication in 1931, of figures obtained for gas tensions in the tissues; he has dealt with the same subject in later articles in 1932.[(7)(13)] Most of the data are from animals, but a few are given for man, including an observation on a gas depot in the forearm, for which, however, no experimental details are given. Earlier and less accurate observations had been made on dogs by Haggard and Henderson.[(10)] Schott[(12)] followed Campbell's technique with rabbits. Observations on the gas tensions in human subcutaneous tissues by Campbell,[(6)] Ellman and Taylor,[(9)] Bazett and Sribyatta,[(2)] and Meyer[(14)] are so inconsistent and at variance with later findings as to be of little value. The findings by Seevers[(11)] (averages from five normal subjects) show a comparatively high carbon dioxide tension (41 to 50 millimetres of mercury) and a low oxygen tension (15 to 27 millimetres of mercury); these are somewhat similar to the results given by Campbell[(6)] for the subcutaneous site in lower mammals (carbon dioxide tension 46 to 52 millimetres of mercury, and oxygen tension 20 to 27 millimetres of mercury). Observations by indirect methods were made by Krogh[(5)] and Verzár.[(15)]

Campbell[(6)] found that the average subcutaneous carbon dioxide tension in cats was greater by 12 millimetres of mercury than the average alveolar and arterial carbon dioxide tensions, which were possibly under-estimated. Bazett and Sribyatta[(2)] found close connexion between carbon dioxide tensions in human subcutaneous tissues and in venous blood, and a more distant connexion for oxygen tensions.

The considerable variation in the figures given by different observers for normal gas tensions in the tissues is due partly, no doubt, to the period of tissue reaction to injected gas, at either an initial or a subsequent injection. The duration of this period has been given variously. Sometimes the tissue reaction appears to have been neglected entirely or confused with the reaching of equilibrium between injected gases and the surrounding tissues. In the present investigations it was found that the duration of the tissue reaction and the period for the reaching of gaseous equilibrium were very variable. Observations on normal gas tensions in the tissues, therefore, should be made as long as possible after both the initial injections and maintenance injections. Several samples should be taken so as to include the range of normal variations, and there must be no continued fall of oxygen tension in successive samples. Physiological variations, which may be caused by changes in muscular activity and temperature and by fear, must be avoided.

To obtain satisfactory normal figures for gas tensions in the tissues considerable experience is necessary both in practical technique and in the behaviour of gas tensions in the tissues under the conditions of observation.

Present Findings on Gas Tensions in the Tissues of Normal Resting Rabbits.

Table I shows a summary of findings concerning gas tensions in the subcutaneous tissues and the peritoneal cavity from 25 normal rabbits.

TABLE I.

Observation.	Site of Depot.	Gas Tensions in the Tissues. (Millimetres of Mercury.)	
		Carbon Dioxide.	Oxygen.
Range of all observations	Subcutaneous. Peritoneal.	40–63 41–61	8–28 14–42
Range of individual averages	Subcutaneous. Peritoneal.	45–55 45–55	15–24 20–40
Average of all observations	Subcutaneous. Peritoneal.	51 49	19 31

These figures are similar to those given by Campbell;[(6)] but the carbon dioxide tensions are somewhat higher and the oxygen tensions lower in the present series. As shown by Campbell, the oxygen tension in the peritoneum is usually slightly but definitely higher than under the skin. Explanation of these phenomena has been suggested in an earlier article.[(3)]

Variations from Time to Time of Gas Tensions in the Tissues.

Variations occur not only amongst different individuals and amongst different tissues, as Campbell has shown, but also to a surprising degree in any tissue from time to time at rest. Hourly samples from subcutaneous or peritoneal depots frequently show variations of carbon dioxide and oxygen tensions of two or three millimetres of mercury or more, under apparently constant conditions. The variations from day to day in individual animals of the series are summarized in Table II.

Sometimes gas tensions in a tissue were found to remain fairly constant for some hours or on two or three successive days; but when observations were continued, considerable variations always occurred.

Under normal resting conditions the gas tensions in a tissue show variations from day to day covering a range of about 10 millimetres of mercury, so that it is better to define gas tensions of a tissue as a normal range rather than as an artificial average. The magnitude of this

[1] Work done with the aid of a grant from the National Health and Medical Research Council.

normal range is better appreciated when it is expressed in terms of the average gas tensions. The average subcutaneous oxygen tension in the series was 19 millimetres of mercury and the corresponding average range was eight millimetres of mercury, or 42% of the average; a maximum of 60% was noted in one rabbit. Any statement concerning the constancy of gas tensions in the tissues must be accepted with reserve.

TABLE II.

Observation.	Site of Depot.	Variations in Gas Tensions in the Tissues. (Millimetres of Mercury).	
		Carbon Dioxide.	Oxygen.
Range of individual daily variations	Subcutaneous.	5–16	5–13
	Peritoneal.	7–15	3–19
Average daily range ..	Subcutaneous.	11	8
	Peritoneal.	11	9

No satisfactory explanation of normal variations of gas tensions in a tissue presents itself. Gas tensions in the respiratory cycle are comparatively constant, and there is no reason to suppose causative changes in general or local metabolism. Indeed it is difficult, even by severe experimental alterations of metabolism, to cause changes in the gas tensions of a tissue approximating the fluctuations observed at rest. No relation could be found between daily variations of gas tensions in the tissues and daily variations of rectal temperature, barometric pressure or room temperature. Rabbits kept in a constant temperature room sometimes showed a tendency to steadiness of gas tensions in the tissues (especially for peritoneal oxygen tension), but major variations still occurred. Excitement can cause fairly rapid changes in the tissues; but this cannot be accepted as an adequate cause, since samples can be withdrawn from a subcutaneous depot in a few seconds with no disturbance to the animal.

It is difficult to imagine that normal variations occur "spontaneously" and independently of tissue metabolism, or that tissue metabolism and the local circulation fluctuate to an appreciable extent at rest. For lack of a known cause an unknown factor must be supposed to account for normal variations of the gas tensions in a tissue, acting either directly on the gas tensions or indirectly through tissue metabolism and the local circulation.

Present Findings on the Gas Tensions in the Tissues of Normal Resting Human Subjects.

In normal human subjects the gas depot method is readily applicable only to subcutaneous tissues. In these investigations the gas tensions of subcutaneous tissues have been correlated with those of the respiratory cycle. Gas tensions in alveolar air were estimated by the Haldane-Priestley method. The carbon dioxide tension of mixed venous blood was found by means of intermittent rebreathing, and the method gave satisfactory results. In a few cases the estimation of oxygen tension in mixed venous blood (a notoriously difficult procedure) was made by the method of Burwell and Robinson.[2]

Observations made on eight gas depots in different sites of the trunk and limbs of a normal subject showed that the gas tensions of subcutaneous tissues are the same all over the body. Of the eight depots, all except one, on which observations were too few for accuracy, showed average carbon dioxide tensions between 45 and 52 millimetres of mercury and average oxygen tensions between 12 and 18 millimetres of mercury.

Table III shows the results of observations on twelve healthy young adult subjects.

The figures given for average gas tensions in the tissues show a somewhat higher carbon dioxide tension and a lower oxygen tension than those given by Seevers[3a] (carbon dioxide tension averaging 45 millimetres of mercury and oxygen tension 22 millimetres of mercury), and they differ greatly from figures for human subjects given by other observers.

The results of human observations are superficially similar to those for subcutaneous gas tensions in rabbits (Table I). The carbon dioxide tensions in both rabbits and human subjects correspond closely in the range of averages, this being 41 to 53 millimetres of mercury in man and 45 to 55 millimetres of mercury in rabbits. The distribution of the individuals of the two series according to the carbon dioxide tension ranges is also similar. As regards oxygen tension, however, analysis shows that, compared with rabbits, human subjects have a pronounced tendency to lower oxygen tensions together with considerably greater variations both for the whole series and for individual averages. For instance, the average subcutaneous oxygen tensions of 50% of the human subjects were lower than the average subcutaneous oxygen tensions of any of the rabbits, whereas the total range of averages for the 12 human subjects (8 to 26 millimetres of mercury) was twice the corresponding range for 25 rabbits (15 to 24 millimetres of mercury).

Variations from Time to Time of Gas Tensions in the Tissues.

Variations of gas tensions from time to time in human subcutaneous tissues could not be investigated as thoroughly as in rabbits. These variations, however, were found to be in all respects similar to those already discussed in animals, as is shown in Table IV. The data were taken from a normal human subject at rest.

TABLE III.

Gas Tensions in Subcutaneous Tissues. (Millimetres of Mercury.)				Gas Tensions in Respiratory Cycle. (Millimetres of Mercury.)				Basal Metabolic Rate; Variation from Normal. (Percentage.)	Subcutaneous Tissues.
Carbon Dioxide Range.	Oxygen Range.	Averages.		Alveolar.		Mixed Venous Blood.			
		Carbon Dioxide.	Oxygen.	Carbon Dioxide.	Oxygen.	Carbon Dioxide.	Oxygen.		
42–60	4–25	49	15	43	94	49	38	+2	Thin.
41–54	20–29	46	24	41	106	46	41	±0	Moderate.
39–54	9–14	50	13	41	98	45			Thin.
40–51	18–26	46	23	43	91	47			Moderate.
48–56	8–20	50	12	38	108	47			Thin.
36–48	12–17	41	14	34	106	42			Thin.
50–58	20–30	53	26	42	102	51	38	−5	Moderate.
44–55	5–15	49	9	40	99	48			Thin.
39–49	15–28	45	21	39	98	45	43	+3	Moderate.
43–55	4–12	50	8	42	95	48	39	−4	Thin.
40–58	10–21	47	16	40	100	47			Thin.
42–52	5–19	46	11	39	94	45	42	+6	Thin.
36–60[1]	4–30[1]	41–53[2] (48)[3]	8–26[2] (16)[3]	34–43[2] (40)[3]	91–106[2] (99)[3]	42–51[2] (47)[3]	38–43[2] (40)[3]		

[1] Showing total range. [2] Showing range of averages. [3] Averages for the series.

TABLE IV.

Time.		Subcutaneous Tissue Gas Tensions. (Millimetres of Mercury.)		Alveolar Air Gas Tension. (Millimetres of Mercury.)		Venous Carbon Dioxide Tension. (Millimetres of Mercury.)
Day.	Hour.	Carbon Dioxide.	Oxygen.	Carbon Dioxide.	Oxygen.	
Tenth	9 a.m.	44	15	41	97	47
Eleventh	9 a.m.	49	19	41	99	48
	11 a.m.	44	22	42	95	48
	1 p.m.	47	20	43	93	48
	3 p.m.	51	16	41	96	48
	5 p.m.	55	18	42	98	47
	7 p.m.	46	17[1]	41	95	47
Twelfth	9 a.m.	51	15	41	99	49
Thirteenth	9 a.m.	51	19	43	96	49
Fourteenth	9 a.m.	46	16	40	96	47
Fifteenth	9 a.m.	52	18	41	99	48
Sixteenth	9 a.m.	47	24[1]	40	93	46

[1] Small specimens.

Technical Consideration of Observations on Human Subjects.

Observations on gas tensions in human subcutaneous tissues must be interpreted critically, with due regard to the sources of error mentioned in this and an earlier article.[12] Many samples of doubtful reliability must be discarded. Most errors give a slight lowering of carbon dioxide tension and a pronounced increase of oxygen tension. Any inaccuracies in the carbon dioxide tensions recorded in the present series are probably negligible. The true upper limit of subcutaneous oxygen tension may be slightly lower than that recorded (30 millimetres of mercury). Very low oxygen tensions (four to eight millimetres of mercury) were observed frequently in tissues with a minimum of thickening and were calculated from reliable specimens. The effect, if any, of the presence of much subcutaneous fat could not be determined, since it was found impossible to make accurate estimations in such tissues. Subcutaneous fat may be the cause of lower subcutaneous oxygen tensions in human subjects than in rabbits.

Normal Carbon Dioxide Tensions in Human Tissues.

Intracellular Carbon Dioxide Tensions.—Owing to the great diffusibility of carbon dioxide in the tissues, the pressure gradient of this gas between tissue fluid and protoplasm is probably negligible.[12] The extracellular carbon dioxide tensions as measured by the gas depot method may be regarded, therefore, as identical with the intracellular carbon dioxide tensions.

Carbon Dioxide Tensions Throughout the Organism.—There is a pronounced uniformity in the range of carbon dioxide tensions (35 to 60 millimetres of mercury) in various warm-blooded species and in different sites in the rabbit (Campbell[10][13] and Table I) and in the subcutaneous tissues (Table III) and other sites[10] in man. Differences may occur in carbon dioxide tensions amongst different individuals (Table III) and amongst different tissues (Table I), but these are not of a gross nature. In general, a lower carbon dioxide tension is to be expected in tissues with permanently high metabolic activity due to an accompanying rich blood supply.

The uniformity of carbon dioxide tensions noted is dependent on three factors: the sensitivity of the respiratory centre to carbon dioxide tension, the high rate of diffusion of carbon dioxide through tissues and tissue fluid, and the local vasodilatation effect of carbon dioxide in the tissues. The respiratory centre so regulates respiration that arterial blood supplied to the tissues has a constant carbon dioxide tension. Because carbon dioxide is very diffusible in the tissues, the carbon dioxide tension of arterial blood sets the lowest limit to carbon dioxide tension in the tissues. The high diffusibility of carbon dioxide and the local vasodilator action of this gas in the tissues together set the upper limit of carbon dioxide tension in the tissues at rest at a level not far above the corresponding tension of mixed venous blood. The observed limits of carbon dioxide tension in human subcutaneous tissues (35 to 60 millimetres of mercury) agree well with these theoretical considerations. There can be little doubt, therefore, that the carbon dioxide tension in any tissue of the body at rest lies between about 35 and 60 millimetres of mercury, and that from time to time the tension will vary widely between those limits, but mostly between 41 and 53 millimetres of mercury (range of averages, Table III).

Normal Oxygen Tensions in Human Tissues.

Intracellular Oxygen Tensions.—The finding of very low extracellular oxygen tensions (four to eight millimetres of mercury) in human subcutaneous tissues indicates that there can be no great oxygen tension gradient between tissue fluid and protoplasm. Since, however, the diffusibility of oxygen in the tissues is low, this tension gradient may be appreciable, up to four millimetres of mercury. The intracellular oxygen tension range in tissues may thus be lower by a few millimetres of mercury than the range observed by the gas depot method. The observed range, however, is so great (4 to 30 millimetres of mercury) that the possible slight correction is of little significance.

Tissue Metabolism at Zero Oxygen Tension.—In the case of the lowest observed extracellular oxygen tension (four millimetres of mercury) the intracellular oxygen tension of those cells furthest from functioning capillaries must be nearly if not quite zero. This does not necessarily imply the presence at times of an anaerobic type of metabolism, nor even the cessation of aerobic metabolism, although tissue activity and compensatory changes in oxygen tension usually rise and fall together. The oxygen tension in a tissue must be taken in conjunction with the corresponding tension in the local tissue capillaries to give the oxygen tension gradient. This in turn is only one of the factors concerned in the volumetric transference of oxygen from capillaries to tissue cells, on which oxidative metabolism is dependent. A low oxygen tension in the tissues permits the possibility of a high oxygen tension gradient from capillaries to cells, facilitating the transference of oxygen.

It is probable that the presence of a very low oxygen tension in the tissues signifies that oxygen transference is so adjusted as to supply just the minimum requirements of the tissues, the whole of the available pressure gradient being used.

Although normal human tissues at times undoubtedly exist at almost zero oxygen tension, usually the oxygen tension is higher. Further reference to this more common state will be made in discussion of the relation of gas tensions in the tissues to the respiratory cycle.

Oxygen Tensions Throughout the Organism.—Oxygen tension ranges show little uniformity in different tissue sites in the rabbit,[10] in subcutaneous tissues of different human subjects (Table III), and amongst different rabbits of the present series. The ranges, indeed, frequently do not even overlap, which is an uncommon finding for carbon dioxide tension.

Contrasted with carbon dioxide, oxygen exerts little effect on the respiratory centre and is 25 times less diffusible in tissues and tissue fluid; perhaps oxygen lack causes less local vasodilatation in the tissues than does carbon dioxide accumulation. These differences, no doubt, partly account for the comparative lack of uniformity of oxygen tension ranges in the tissues. These ranges may vary, theoretically, from arterial oxygen tension (about 80 millimetres of mercury) down to zero. It would be expected that the greater the normal metabolic activity of a tissue, the higher would be its oxygen tension, owing to its permanently rich blood supply.[10] Thus active tissues, such as cardiac muscle, would have a higher oxygen tension than less active tissues, such as subcutaneous tissue at rest.

Table III shows that the oxygen tension in human subcutaneous tissues varies between about zero and 30 millimetres of mercury. Oxygen tensions no doubt vary as much amongst different tissues in human subjects as in rabbits,[9] so that the total range of oxygen tensions in human tissues at rest is probably at least 0 to 50 millimetres of mercury. It is improbable that any normal tissue at rest has an oxygen tension much above 50 millimetres of mercury, since the oxygen tension of mixed venous blood is only about 40 millimetres of mercury at rest.

Gas Tensions in the Tissues in Relation to the Respiratory Cycle.

Comparable figures for gas tensions in the tissues and in the respiratory cycle are given in Tables III and IV. All the figures for alveolar air and venous blood are within normal limits.[11] Oxygen tension in arterial blood is significantly lower (by 10 to 20 millimetres of mercury) than in alveolar air,[12] so that the average alveolar oxygen tension of 99 millimetres of mercury may be regarded as representing 80 to 90 millimetres of mercury arterial oxygen tension.

The close correspondence of carbon dioxide tensions in the tissues and in venous blood is in accord with Krogh's prediction from his microscopic observations on capillaries in the tissues and from the high diffusibility of carbon dioxide in the tissues.[13] It has already been shown that the arterial carbon dioxide tension is the lowest limit for carbon dioxide tension in the tissues. The upper limit of normal carbon dioxide tension in the tissues (60 millimetres of mercury) is well above the carbon dioxide tension of mixed venous blood. This indicates that the carbon dioxide tension in tissues and local venous blood varies from time to time and from tissue to tissue, rather than that the carbon dioxide tension in a tissue is at any time appreciably higher than in the local venous blood. It may be assumed that the carbon dioxide tension of mixed venous blood is an accurate indication of the average carbon dioxide tension of all the tissues of the body.

Oxygen tension in mixed venous blood at rest (about 40 millimetres of mercury) is on the average about 24 millimetres of mercury higher than in human subcutaneous tissues (averaging about 16 millimetres of mercury). The oxygen tension in venous blood which is purely local to subcutaneous tissues is not known; but the oxygen tension gradient between the local venous blood and subcutaneous tissues is undoubtedly considerable. For purposes of comparison with the oxygen tension of mixed venous blood an average oxygen tension throughout the tissues of the body of about 25 millimetres of mercury may be assumed; this represents the mean of the probable total range (0 to 50 millimetres of mercury).

The relation of the gas tensions in the respiratory cycle to those in the atmosphere and in the tissues may be summarized by the following scheme. Average gas tensions are shown, the figures representing millimetres of mercury partial pressure. The gas tensions in subcutaneous tissues are shown in relation to those of mixed venous blood, since direct observations have been made in these sites, although they are not strictly comparable. Substitution of the mean figure of 25 millimetres of mercury oxygen tension for 16 millimetres of mercury subcutaneous oxygen tension would give a fair representation of the relations of tissues in general to the respiratory cycle. Arterial oxygen tension may be a little higher than is shown in the diagram.

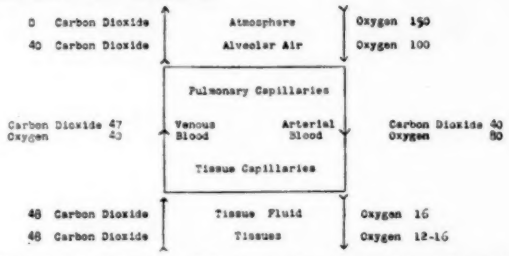

Diagram showing in millimetres of mercury the differences in oxygen and carbon dioxide tensions in different tissues.

The Significance of the Oxygen Tension Gradient.

The high oxygen tension gradient between capillary blood and the tissues at rest is of great significance. During increased tissue activity the slowing of the rate of capillary blood flow,[22] apart from other compensatory mechanisms, will enable the tissues to withdraw more oxygen from capillary blood. This would not be possible if the oxygen tension in resting tissues were not lower than oxygen tension in capillary blood.

The resting oxygen tension gradient is not the maximum available when tissue activity increases. There is a tendency to both a concurrent fall of oxygen tension in the tissues, due to accelerated oxygen consumption, and an increase in capillary blood oxygen tension, due to the relaxation of arterioles. The available "head of pressure" for oxygen supply is thus greatly magnified. The opening up of the capillary network during tissue activity also increases vastly the area available for oxygen diffusion and lessens the average distance between capillaries and cells.[13] Not only is diffusion facilitated, but there is more blood available in the capillaries from which oxygen may be withdrawn.

In tissues at rest the high oxygen tension gradient between capillary blood and cells no doubt represents a potential reserve for increased oxygen supply in conjunction with the reserve capillary network. Similar reserve mechanisms are present at the other pole of the respiratory cycle, in the pulmonary capillaries and alveolar air.

Summary.

1. There is great variability in the tensions of gases, particularly that of oxygen, in the tissues of normal resting subjects. Variations amongst different tissues may be accounted for by differences of metabolic activity; but variations from time to time and amongst normal individuals at rest are of unknown origin.

2. The ranges of carbon dioxide tension in the tissues are more uniform than those of oxygen tension. Carbon dioxide tension varies from about 35 or 40 millimetres of mercury (arterial carbon dioxide tension) to about 60 millimetres of mercury, averaging about the level of the carbon dioxide tension in mixed venous blood.

3. Oxygen tension ranges are very variable. In human subcutaneous tissues the range is about 0 to 30 millimetres of mercury, and the tension is often observed to be below 10 millimetres of mercury. Oxygen tensions throughout resting tissues are probably mostly well below the oxygen tension of mixed venous blood.

4. Occasional oxygen tensions approaching zero in resting tissues indicate a low oxidative metabolism with a high oxygen pressure gradient between capillary blood and tissues.

5. The normal high oxygen tension gradient from capillaries to resting tissues represents a potential oxygen reserve for increased metabolic activity.

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Reports of Cases.

LEPROSY WITH RAPID PROGRESS.

By J. H. BLACKBURN, M.A., M.D., L.R.C.P. (Edinburgh), F.A.C.C.P.,
Visiting Government Medical Officer, Woorabinda Aboriginal Settlement, Queensland.

G.D., A MARRIED aboriginal woman, aged twenty-nine years, had an indurated anæsthetic patch on the right arm in August, 1939, when she was first examined; in May, 1940, she had indurated anæsthetic patches on the right side of the body in the following sites: above the eyebrow, in the arm, in the buttock and above the knee and ankle. Examination of all the smears gave positive results. As far as is known, the patient has otherwise enjoyed a measure of excellent health. She was in the habit a few years ago of playing with a baby, three members of whose family were lepers.

Comment.

The case is published on account of the rapid progress, as it is well known that leprosy as a general rule takes from five to forty years to become active.

Acknowledgement.

I wish to thank Sir Raphael W. Cilento, the Director-General of Health and Medical Services, Queensland, for permission to publish the case, and Dr. B. R. V. Forbes, Medical Officer-in-Charge of the Commonwealth Health Laboratory, Quay Street, Rockhampton, for kindly staining the slides and examining them.

Reviews.

MEDICAL TREATMENT.

THE second edition of "Vade Mecum of Medical Treatment", by W. Gordon Sears, provides a valuable addition to the reference library on current methods of medical treatment, and it is published in a convenient size with cloth cover.[1] The arrangement of the contents is alphabetical, according to the diseases, and there is an alphabetical index at the end of the book.

The subject matter is written in a very practical style, many important diagnostic points being given. It also includes reference to the use of many proprietary preparations which have earned a reputation as the result of experience.

The book is liberally supplied with well-tried and trusted prescriptions, which should prove very useful to the student and busy general practitioner alike.

It is interesting to find that the time-honoured Latin, which has been used in prescriptions, is gradually being replaced by English to such a degree that the Latin abbreviations have even lost their true reference. For example, in connexion with chronic dacryocystitis on page 122 we read:

℞

Zinci Sulph.	gr. 2.
Liq. Hydrarg. Perchlor.	dr. 1.
Aquæ Lauroceracis	ad 1 oz.

1 drop in affected eye, *t.d.s.*

The *t.d.s.* obviously does not mean "to be taken three times a day", but rather *"t.i.d."*, three times in a day.

There is an interesting brief appendix dealing with the use of sulphanilamide and allied drugs. Many useful tables on drugs, measures, calorific values of foodstuffs *et cetera* appear at the end of the book.

As a publication bearing the title of "Vade Mecum of Medical Treatment" this book should be appreciated by practitioners who require some quick reference to general subjects.

LEPROSY.

THE second edition of "Leprosy", by Sir Leonard Rogers and Ernest Muir, is an encouraging book.[2] That rigid compulsory isolation was defeating its own ends by causing concealment of early cases has long been realized. Here the futility of this system is thoroughly demonstrated and the modern alternative is described, with proofs of its efficiency. The chief points are: home isolation under strict supervision or segregation in agricultural colonies, with the minimum of possible compulsion, for advanced and infectious cases; the dissemination of the knowledge that vigorous treatment in the early stages can rapidly render patients uninfectious and can offer a reasonable hope of cure; treatment at out-patient clinics of uninfectious patients; and removal of the more highly susceptible children from possible risk of infection. The lesson is hammered home in chapter after chapter; but the arrangement of the subject matter is so good that there is no sense of wearisome repetition.

The sections on ætiology, classification, clinical signs and pathology are exhaustive, and the well-produced illustrations are very helpful. Treatment is well outlined. The means of diagnosis are fully described. The only serious drawback is that where very young children are concerned, the clinical methods are inapplicable, and the Matsuda test is unreliable; in doubtful cases, apparently, reliance can be placed only on repeated bacteriological examinations. A few hints as to the procedure to be adopted in dealing with suspected early leprosy in a white child, for instance, without unduly alarming or antagonizing the parents, would have been welcome.

The production of the book is good. Style has been generally sacrificed in favour of conciseness, and this method can only be condemned in those places where it has led to confused constructions or definite errors; there are too many of these.

This is a useful and valuable book, thoroughly justifying its existence.

[1] "Vade Mecum of Medical Treatment", by W. G. Sears, M.D., M.R.C.P.; Second Edition; 1939. London: Edward Arnold and Company. Crown 8vo, pp. 383. Price: 10s. 6d. net.

[2] "Leprosy", by Sir L. Rogers, K.C.S.I., C.I.E., M.D., F.R.C.P., F.R.C.S., F.R.S., I.M.S., and E. Muir, C.I.E., M.D., F.R.C.S.; Second Edition; 1940. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 272, with illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 2, 1940.

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EMPIRICISM IN MEDICINE.

THE wise clinician spends more time trying to understand the cause of disease than he does in seeking to make himself familiar with the drugs reputed to cure it. In other words, he is less concerned with medicaments than he is with the nature of the infective or other agent of disease, how it gains access to or is enabled to have an effect on the body, and what changes it produces in the body tissues. He realizes to begin with that once he knows what the cause is he may be able to prevent the occurrence of the disease; he also knows that if he is able to put his finger on the cause, he has some chance of elaborating a specific cure; moreover, he can be quite certain, though he may not always care to admit it, that if no cause has been discovered, all his remedies are somewhat like a shot in the dark, brought into use because someone has told him they are effective or because in apparently similar cases their exhibition has been followed by recovery. If we exclude preventive measures, the most desirable form of treatment, we know that the response of the individual, even to specific treatment, is not predictable. Always there is the *vis medicatrix naturæ*, unexpected in its effects and at times almost capricious. Every clinician can tell of patients, apparently at death's door, who, according to accepted standards, have had no hope of recovery and who have confounded the prophets by return to health and strength; while others, the recipients of what is regarded as a light infection, have succumbed without putting up a fight. The clinician (we have called him wise) who habitually looks for the cause of disease that he may remove or overcome it, is often disappointed. There are so many mysteries waiting to be solved—the causes of so many diseases have yet to be discovered—that our wise clinician has not seldom to resort to symptomatic or empirical methods. He may then be confused by the unwary with his not so wise brother who delights in the number, the elegance, nay, the fashion, of his medicaments. The confusion will not perturb him; but it will be a pity if the unworthy is regarded as wise and is confirmed in a course that cannot bring credit to the profession of medicine. In these circumstances it may be useful to consider the nature of empiricism, its place in medicine and the safeguards that are needed when it is used.

Empiricism is a branch of philosophy according to which truth can be discovered by repeated observation and experiment. Empiricism in medicine, we are told by Clifford Allbutt in his FitzPatrick Lectures delivered more than thirty years ago, arose as a reaction to the extravagances of the dogmatists. Allbutt showed that certain notions in medicine, particularly the humoral doctrines, became crystallized out in exaggerated shape and that principles and even collections of words became detached as abstract dogmas and terms, and gained acceptance as forces or entities. To these tendencies were added influences from occultism, astrology, demonology and other extravagances. It is not surprising that the cleavage between the dogmatists and empirics became increasingly wide and that, as Allbutt puts it, in the fury of their disputes and repartees the points of difference were forced into exorbitant contrasts. This is a characteristic human failing and will be found cropping up in every age. The dogmatists were represented as recognizing no facts and the empirics no reasons. Allbutt expressed the view that empiricism was a valuable discipline because it based itself on action; it was indeed probably necessary, and it certainly marked an epoch in medical thought and endeavour. Celsus represented the empirics as relying first of all on the chance observation of happy results, but also on deliberate experiment and the systematic collection of facts. Allbutt credited them with common sense; he pointed out that they believed in induction, though they did not apprehend the inductive method in its completeness; and, in describing their faults, said that they were unaware of the fallacy of *enumeratio simplex*, had no criteria and in treatment would be guided by the uppermost signs. Such was the empiricism of Greek medicine. There is no doubt that the more rational empirics of the day, combining with their superior empiricism the clinical descriptions that had been handed down to them by Hippocrates and his followers, were in advance of others who knew nothing of their doctrines, and of those who practised them in an imperfect form. If an analogous statement is made about present-time conditions, when the progress of pathology and the development of the ancillary sciences have revealed the nature of many diseases, it is clear that those who cleave to empirical methods have been superseded by those who try to apply modern scientific methods in their approach to disease.

Though we are practically back again at the point from which we started, we have seen that empiricism has not been completely outmoded. Unfortunately the term empiric is most often applied to those who carry empirical methods to extremes. Just as in the Grecian days a debased empiricism, owing to its recognition only of the coarser phenomena, was little removed from folk medicine, so today there is a base empiricism which is little better than guesswork or rule of thumb. What must be remembered is that as long as a clinician is ignorant of how a remedy acts in any disease, his treatment is purely

empirical. The use of empiricism is always a confession of ignorance; but a sharp distinction must be drawn between ignorance that is shared by the whole medical profession and the culpable ignorance of debased empiricism which is likely to be attributed to even unworthier origins. There is little to be said, for example, in favour of a practitioner who in the presence of an obscure febrile illness does not pursue every method of investigation, but gives a sulphanilamide compound in case there may be a streptococcus about; or of another who, finding a patient slow to recover from an illness, diagnoses "slight anæmia" and gives expensive intramuscular injections of liver extract without a preliminary complete blood examination. On the other hand, we can have nothing but admiration for a practitioner who knows that his patient is suffering from a self-limiting infection and who refuses to yield to the importunities of patient or friend to prescribe some much-vaunted remedy because progress is halting and recovery slow. Empiricism still finds, and will continue to find, a place in the practice of medicine; but he who employs empirical methods must be continually on his guard lest by paying attention to the phenomena staring him in the face, he neglects others less obvious that may be of significance; further, he should not look on his premises as entirely reliable or complete, nor should he regard his conclusions as final. Allbutt spoke truly when he said that the empiric did not see that the human mind could not content itself with perpetual suspense, but, in default of some chain of reason, would betake itself to analogies which were usually superficial and false. He added that the empiric did not see the influence of the sciences in the training of the mind as an instrument to accuracy and method. The empiric must walk as a blind man, feeling his way cautiously with every step and fearful lest he should fall.

Current Comment.

SEPTICÆMIA.

A USEFUL "critical review" on the subject of septicæmia comes from the pen of L. E. H. Whitby.[1] Septicæmia is a condition about which not much that is new can be written except in the matter of treatment, where chemotherapy now overshadows all other methods. The outlook for the patient has changed from what it was, say, thirty years ago, when we were told that acute septicæmia, puerperal or otherwise, if of streptococcal origin, should always be treated by vaccine therapy; and when it was held that the occasional success of antistreptococcic serum depended upon its accidental value as a vaccine. Apart altogether from questions of treatment, a discussion on septicæmia is particularly welcome at the present time, when wounds are inflicted indiscriminately on civilians as well as on members of the defence forces.

Whitby starts by reminding his readers that the disease is often cryptogenic, so that no portal of entry can be discovered, and he adds that virulent and invasive organisms may penetrate an unhealthy mucous membrane, especially in the pharynx, without exciting a local reaction sufficiently characteristic for a clinical examination to differentiate it from any other local inflammation. Although an abrasion of the skin may be almost microscopic, the portal of entry is usually clear or may be surmised with a moderate degree of confidence. Septicæmia may be the terminal condition of an infective disease, and in war wounds, where there is great laceration of tissues and deep structures are involved, the risk of septicæmia is great. The recognition of any local focus is obviously of the greatest importance, for recovery is impossible unless a local focus responsible for the blood infection is "eliminated, localized or rendered incapable of feeding the blood-stream further". This statement has reference to a septicæmia already established. In regard to an infection that has not yet been followed by septicæmia, Whitby points out that its continued localization depends on many factors. "If it is a case of wounding, the chance of invasion is influenced by the amount and the nature of the tissue wounded, for some tissues have more power of localizing an infection than have others. The immediate protection of the body against septicæmia in any case of wounding is the reaction of inflammation which consists in the outpouring of lymph and phagocytic blood-cells at the site of the wound. The longer that this reaction can keep a local bacterial implant from multiplying or invading the blood-stream, the greater is the body's opportunity for exerting its more permanent protective action, namely, the production of granulation tissue." Surgeons have learned, sometimes after long experience, the desirability of disturbing a wound as infrequently as possible for the changing of a dressing, and there is no doubt that fixation by plaster of Paris, as practised today, will often allow granulation tissue to be formed and minimize the risk of septicæmia. The final stages in recovery from any septicæmia depend, as Whitby states, upon the ability of the body to produce antibacterial substances in sufficient quantity to prevent multiplication of bacteria and to render them susceptible to phagocytic destruction. This is why specific antiserum is used in septicæmia and also why blood transfusions are given; the latter procedure is "believed to supply complement and other substances which are frequently lacking".

Whitby makes useful remarks about the prophylaxis of wounds, particularly those received by medical men making post-mortem examinations; these unfortunately cannot be reproduced owing to lack of space. In discussing surgical principles, he points out that particular infections demand particular treatment and adds that coccal foci of infection are better not incised until pus is laudable. "It should also be borne in mind that the formation of an abscess sometimes serves to localize an infection and . . . to save life." This is easily forgotten, as is the fact that artificial abscesses have sometimes been produced as a method of treatment. In regard to chemotherapy, there is no need to emphasize the giving of a large initial dose of a suitable compound in order to obtain quickly an adequate concentration of the drug in the blood. Difficulty will quite often be experienced in determining the duration of treatment. This is found in all forms of chemotherapy, and quite commonly sulphanilamide drugs are given for too long a period. Whitby thinks that if no action is noticed within ten days it is useless to pursue treatment with that particular drug. For a second course the preparation should be changed. Two points remain. The first is that because chemotherapy is known to be effective there is a tendency to rely on it alone. "Pus requires surgical treatment." The second point is that chemotherapy is not for the timid who are "apt to prescribe small and therefore ineffective doses".

[1] *The British Journal of Surgery*, July, 1940.

DENTAL CARIES.

R. F. SOGNNAES AND R. L. WHITE[1] have made a study of the factors influencing the incidence of dental caries in a group of American children, and have taken into consideration the general structure of the mouth, the state of general health and the habits of life of the children. From a large dental clinic two groups of children were selected, purely on a basis of incidence of dental caries. Group 1 consisted of the clinic patients with the lowest and Group 2 the highest incidence of dental decay.

[1] *American Journal of Diseases of Children*, August, 1940.

Deformities of oral structure were common in Group 2; of these, one-third had malformations interfering with the proper occlusion of the jaws, and more than one-half had a high narrow palate. The incidence of initial lesions of the white enamel, showing as white opaque lines or spots and known as "precaries", was found to be much higher in Group 2. In deciduous teeth no children of Group 1 and 55% of Group 2 showed precaries. In permanent teeth the figures were 14% and 93% respectively. The gums were unhealthy and inflamed in 61% of Group 2, but in only 15% of Group 1. The use of the tooth-brush, however, was about the same in both groups; 70% of each claimed to clean their teeth regularly. Staining of the teeth, usually an evidence of insufficient cleaning, was indeed slightly higher in Group 1.

The mothers of Group 1 were on the average four years younger at the birth of these particular children, and the families were smaller by a little more than one child per family than in Group 2. The mothers of Group 1 had also previously borne fewer children.

A general assessment of the factors of significance acting during the pregnancy preceding the birth of these children was made. The factors considered were the age of the mother, the extent of medical supervision, the history of general health and hygiene, the existence and duration of nausea and vomiting, and the character of the diet. In the first group 79% of the mothers were rated as "excellent" or "good", but in the second group only 28% achieved this rating.

A similar assessment was made of the health and progress of the children during the first year of life. The factors considered were extent of medical supervision, ease of adjustment to food, the existence and duration of breast feeding, the diet of the mother during lactation, the age at which cod liver oil and orange juice were added to the diet, the character of the remainder of the diet, and the history of general health and progress. A rating of good or excellent was given to 86% of the children of Group 1, but to only 28% of the children of Group 2. Statements regarding the baby's good nature and ease of adaptation to routine were frequently made in the histories of the Group 1 children. Breast feeding was much more frequently used and for longer periods in Group 1, a factor of great importance, for breast milk is considered unique in its ability to meet the requirements of developing human tissues, and breast suckling is of the utmost importance in determining the proper formation of the dental arch and the muscles of the throat and jaws.

The common infectious diseases of childhood seemed to have been as prevalent in one group as the other. A general physical examination revealed evidence of old rickets in 50% of Group 2, but in none of Group 1. Obesity, poor posture and nervousness were also more common in Group 2. Hypertrophied tonsils were found in about the same number in each group.

The present diet of the children was carefully examined, with particular reference to the quantity of "protective foods", that is, foods valuable for their mineral and vitamin content, such as milk products, fruit, vegetables, eggs and whole-grain cereal products. The diets of 57% of Group 1 and 11% of Group 2 were regarded as adequate in this respect. Cod liver oil had been given to 57% of Group 1 and to 17% of Group 2 during the winter preceding the study. During previous winters there had been little difference between the numbers in each group taking cod liver oil. The consumption of sweets and cakes between meals was appreciably higher and with less effort at parental control in Group 2. Perhaps as a consequence the appetite was classed as poor or capricious in 61% of Group 2 as against 14% of Group 1. The consumption of "adhesive carbohydrates", such as cakes, pastry, white bread, jam and refined cereals was regarded as excessive in 14% of Group 1 and 67% of Group 2, figures strikingly the reverse of the consumption of "protective foods".

Despite these striking differences in diet, control and general health, there was no significant difference between the social and economic status of the families. There was, however, a noteworthy difference in the interest and intelligence with which the money was administered. This same intelligence found expression in a general awareness of health responsibility in the mothers of Group 1. An assessment was made of the factors indicative of such general health awareness, namely, the child's habitual amount of sleep, his personal cleanliness, the household routine, the mother's use of community health resources for public health education, the presence or absence of "food finickiness" on the part of the parents, the emotional stability of the home, and the character of the child management. A rating of good or excellent was given to 86% of Group 1, but to only 45% of Group 2. This study bears out many of the conclusions that were stated in a leading article in this journal in October, 1939, and we may conclude that it seems that it is our parents who give us good teeth or bad.

DISTURBING INFLUENCE OF GOLF ON THE BLOOD SUGAR.

THERE must be many thousands of medical men whose recreation for one reason or another is golf and who try very hard to acquire proficiency at the game, persisting doggedly despite exasperating failure. These will be grateful to Dr. P. Michael, of Oakland, who has recently shed light on some part at least of the physiology of erratic golf.[1] He selected thirty healthy men between thirty-five and forty-five years of age, whose golf handicaps ranged from ten to twenty-seven strokes, and he determined their blood sugar levels after every alternate hole during afternoon rounds of golf. As far as possible the investigation was made under the normal conditions of play; the players ate their usual luncheons and rested for twenty minutes before setting out; the only exception was that they were not allowed to smoke. He found that between the eleventh and fifteenth holes in twosome matches and between the ninth and fifteenth holes in foursome matches the amount of sugar in the blood was below the fasting level. In each case the blood sugar level was lowest after the thirteenth hole, the average being 0·055%. The play was least accurate during the period of hypoglycæmia, and during this period also some of the players complained of fatigue. These phenomena were more pronounced in the poorer golfers, who, of course, expended more energy in playing the game, and in those who were "highly strung and excitable"; the blood sugar level was less disturbed in the more poised and accomplished golfers. In fact, Michael suggests that there may be a vicious circle: the poorer golfers were the more anxious and they showed more mental and emotional fatigue, which in turn might be expected to impair their play and also, because of the liberation of adrenaline, to produce, after a temporary rise, a fall in the blood sugar level.

Michael makes two suggestions of measures to combat the period of hypoglycæmia: first, a more ketogenic type of luncheon, and second, increased carbohydrate intake in the form of sugar or "candy" near the eighth hole in a foursome and near the ninth in a twosome; and he states that these measures are followed by a decrease in untoward symptoms and on the whole by better scores. However, the imaginative golfer will see at once that Michael has touched only on the fringe of the remedy, and that sweeping changes in the conventions of golf are imminent. In the future the perfect caddy, far from being pained by the note of acerbity in the player's voice and the savage glint in his eye as he calls for a niblick at the eleventh, will politely proffer manna in the shape of a large hard confection to serve the dual purpose of sealing the lips and raising the blood sugar. That excellent institution, the nineteenth hole, will doubtless be advanced in its position and renumbered 8A, and a tempting variety of carbohydrate foods, which we need not specify, will fortify the flagging or fidgety player when he is most in need of refreshment and sustenance.

[1] *The Journal of the American Medical Association*, July 27, 1940.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Sulphathiazole and Sulphapyridine.

HARRISON F. FLIPPIN, LEON J. SCHWARTZ AND S. BRANDT ROSE (*Annals of Internal Medicine*, May, 1940) treated a series of patients suffering from pneumococcic pneumonia with sulphathiazole. They gave all patients an initial dose of three grammes, repeated this in four hours, and then gave one gramme every four hours until the temperature had been normal for forty-eight hours. Some patients were given an intravenous dose of a 5% solution of the sodium salt also. Blood cultures and typing were undertaken, and blood estimations of the concentration of the drug were also made. Control patients were similarly treated with sulphapyridine. The results were compared. In the series treated with sulphathiazole the total mortality rate was 12%; in the series treated with sulphapyridine it was 15%. In the cases in which cure was achieved the critical fall in temperature occurred within forty-eight hours in 88% of the sulphathiazole cases and in 95% of the sulphapyridine cases. The length of stay in hospital of both groups was the same (13·2 days). In a small group of cases in which the drugs had failed, serum therapy was also instituted. These were all serious cases, and eight of the nineteen patients died. The authors believed that serum effected the recovery of several patients who had failed to respond to the drugs. Complications were few and were comparable in the two groups, being 5% in each case. The toxic reactions to the drug were, however, very different. In the sulphapyridine group nausea and vomiting occurred in 84%, and in four cases they necessitated the discontinuance of the drug; ten patients showed microscopic hæmaturia. Blood studies failed to reveal any reduction in hæmoglobin value or in the numbers of red or white cells. In the sulphathiazole group nausea and vomiting were slight and occurred in 34% of cases; they were never of such dimensions as to cause suspension of the drug. Six patients showed microscopic hæmaturia. The authors believed that the drugs were of almost equal efficiency in the treatment of pneumonia, that sulphapyridine appeared to be the faster acting drug, judged by the critical fall in temperature, but that the nausea and vomiting produced by sulphathiazole were much less than in the sulphapyridine group. They felt that serum of the appropriate group was indicated in certain severe cases in which the drugs produced no response within thirty-six to forty-eight hours.

Specific Agents in Soil.

RENÉ J. DUBOS (*Annals of Internal Medicine*, May, 1940) studied the effect of specific agents extracted from soil microorganisms upon experimental bacterial infections. His experiments were based on the belief that organic matter does not accumulate in Nature, but becomes the prey of microorganisms or catalysts which break it down into its ultimate element. He isolated from soil an entirely new sporulating bacillus which hydrolysed the specific capsular substance of the Type III pneumococcus, which was inactive against all other polysaccharides tested. When pneumococci were deprived of their capsules in a medium containing the enzyme, and subcultures were made into medium not containing the enzyme, the cocci again exhibited capsule formation, so that the enzyme did not affect their growth. *In vivo*, however, when mice were injected with virulent Type III pneumococci and enzyme solution, the decapsulation effected by the enzyme allowed phagocytosis to complete their destruction and the mice survived, while all control animals, which did not receive enzyme, died. Monkeys also were used to show the effectiveness of the enzyme, and were protected from ill effect. It was possible, however, to give a dose of organisms of such size that the enzyme effect was not shown. In further proof of his contention, the author incubated pneumococci, streptococci and staphylococci in a soil mixture, in search of a soil flora inimical to the Gram-positive cocci. He isolated an aerobic sporulating bacillus which produced a soluble principle that was extremely toxic to Gram-positive cocci, and further obtained it in pure form by alcohol-ether extraction and acid precipitation. He collected two fractions, in the first of which he named two substances, graminic and graidinic acid, and in the second one substance, gramicidin. These substances were highly bactericidal to pneumococci or streptococci, dilutions of 1×10^{-6} being effective. They were also active against diphtheroid bacilli and staphylococci, but were not at all active against Gram-negative bacilli. *In vivo* tests were also made, the standard being the amount which when injected intraperitoneally would protect mice against 10,000 fatal doses of the organisms. It was found that the substances had to be in contact with the organism; the intravenous injection of test substances, for instance, was of no avail against intraperitoneal injection of organisms. The author compared the action of his enzyme solution with that of antiserum, in that it merely prepared the organism for the action of phagocytes, rendering it susceptible by reason of the loss of its capsule. The gramicidin group, on the other hand, as their name suggests, acted as true antiseptics, inhibiting the growth of organisms in small concentration, and actually killing them in higher concentration. He finally correlated the activity of the bactericidal agent with the staining properties of the organisms against which it was effective, assuming that these were dependent upon structural characteristics. He hoped that these observations would lead to rationalization of chemotherapy by the conjoint work of chemist and bacteriologist.

Malaria Antibodies.

L. T. COGGLESHALL (*The Journal of Experimental Medicine*, July, 1940) continued his experiments with malaria and investigated the occurrence of antibodies in human serum following induced infection with *Plasmodium knowlesi*. Seven Negro patients suffering from general paresis were given therapeutic injections from rhesus monkeys which had been shown to be suffering only from *Plasmodium knowlesi* infection. Varying doses of parasites were used, from 2,500,000 given intramuscularly to 10,000,000 given intravenously, and all patients exhibited typical malarial attacks which terminated spontaneously. The patients were bled at intervals after recovery and the serum was used for protection tests in normal monkeys, for complement fixation tests with saline extracts of *Plasmodium knowlesi* as antigen, and for agglutination tests; but the last were not satisfactory. The results of the protection tests varied a little in the time at which the serum exhibited its maximum effect; but the complement fixation test showed that the titre of the serum was at its maximum at the same time, although the maximum was seldom a high figure. The author discusses the reason for this, and suggests that the time in the life cycle of the parasite at which the test was performed was of importance in the determination of the result, as a young parasite protected by the cell envelope would be less accessible to the action of an immune serum than a mature one which had stretched the cell envelope to its thinnest. Further, the fact that the patients from whom the serum was taken were Negroes influenced the result, as these people are relatively resistant to infection; although they were untreated, they recovered from the infection spontaneously in seven days. The author thought that more susceptible subjects might have produced a more potent antiserum and shown a more specific protective effect. He was satisfied that the experiment proved that antibodies were formed in the serum of human patients following induced infection with *Plasmodium knowlesi*.

The Capsule of the Plague Bacillus.

S. S. SOKHEY (*The Journal of Pathology and Bacteriology*, July, 1940) has demonstrated the capsule of the plague bacillus. The work was founded on the observations of Churchman and Emelianoff, who showed that adequate technical methods could frequently demonstrate the presence of capsules on organisms not generally recognized as possessing them. The method, which is described in full, was applied to the plague bacillus for the first time. Much importance was attached to long immersion in Wright's stain, rinsing with buffer solution, and rapid drying of the preparations. The capsule could be shown in cultures grown at temperatures ranging from 5° to 37° C., either in liquid or on solid media, and in organs taken from mice dead of infection. Capsules were also clearly visible on organisms which were avirulent. This disposed of the suggestion that capsule formation and virulence are associated in this organism.

Encephalomyelitis of Mice.

MAX THEILER AND SVEN GARD (*The Journal of Experimental Medicine*, July, 1940) have studied encephalomyelitis of mice. The two strains under investigation were accidentally isolated during the investigation of yellow fever. The suggestions were made that there were many points of similarity between mouse encephalitis and human poliomyelitis, and that detailed study of the mouse disease might shed light on the human form, for which there was but one laboratory animal, expensive and difficult to obtain. The mouse strains isolated had a much higher morbidity and mortality rate amongst young animals; adult animals, while differing in different groups, were on the whole far less susceptible. The viruses were extensively examined *in vitro* and were found to be of similar size to that of the poliomyelitis virus; they were inactivated by alcohol and precipitated

from suspensions by ammonium sulphate. *In vivo* experiments with large groups of mice showed that both strains produced lesions of the brains and central nervous system when intravenous or intracerebral inoculation was made, but by the intraperitoneal route one strain produced mainly flaccid paralysis of the limbs. Immunity to further infection could be produced in animals which had survived intraperitoneal or intranasal inoculation of virus of homologous type. Immunity was also produced by intracerebral inoculation of test virus in animals which had previously been given intracerebral inoculation of a similar but relatively avirulent virus. The distribution of virus in the central nervous system, viscera and nasal mucosa was tested and the times of their appearance were recorded.

MAX THEILER AND SVEN GARD (*ibidem*) have continued their investigations into the epidemiology of the encephalomyelitis of mice. Their attention was drawn to the fact that while occasional cases occurred in their mouse population, no evidence indicated cantagion from mouse to mouse, and the presence of mice experimentally infected did not lead to the development of immunity in contacts. They felt that a large number of the mice must be protected from the virus in some way. The intestinal contents of the apparently normal mice were collected and investigated, and it was found that young mice, aged up to fifty-three days, harboured a virus in the small and large intestines. When this virus was inoculated into very young mice it caused paralysis and death. Any survivors could not later be infected with the virus of mouse encephalitis. No less than two-thirds of the mouse population harboured this intestinal virus, so that the low incidence of encephalitis could be explained on the theory of protection conferred by its presence. The authors suggest, on hypothetical grounds, that there might have been an increasing development of antibodies in response to infection of the intestinal wall, which made the animals resistant to other infection.

Poliomyelitis Virus in Human Stools.

HOWARD A. HOWE AND DAVID BODIAN (*The Journal of Infectious Diseases*, May-June, 1940) investigated untreated human stools as a source of poliomyelitis virus. Their method was simple and did not require bactericidal methods to be applied to the specimens in which virus was being sought. The stools were emulsified in distilled water and strained, and thick suspensions were sealed in ampoules of 10 cubic centimetres' capacity and subjected to a single cycle of thawing and freezing; they were then resuspended and inoculated intranasally into monkeys, each animal receiving from three to eight inoculations of one cubic centimetre in each nostril. Fourteen specimens were examined, all being taken from human patients showing paralysis. Ten caused paralysis in the animal and typical lesions in the central nervous system. The animals showed no abnormal reaction to the inoculation except in one case, in which there was a febrile reaction after the second intranasal swabbing. The incubation period for the disease varied from nine to sixty-four days; the majority of lesions occurred in the second week. Most animals showed paralysis of the legs or arms, and if not killed and examined tended to functional recovery. All specimens of stool taken within five days of onset of the patient's paralysis gave positive results. The authors were satisfied that the technique was simple and reliable, and caused less intercurrent infection amongst laboratory animals than methods requiring intracerebral inoculation.

JAMES D. TRASK, JOHN R. PAUL AND ALFRED J. VIGNEC (*The Journal of Experimental Medicine*, June, 1940) investigated the poliomyelitic virus in human stools. Specimens were obtained during four epidemics and from widely varying centres and were treated by extraction in ether followed by cold sterile distilled water. All monkeys used were inoculated intraabdominally; in a few cases intracerebral inoculation was added, in some with test solution, in others with sterile 1% starch solution. One hundred and eleven tests were completed in the series, and ten yielded positive results. The first patient yielding virus in the stool had the disease in a mild form and developed into an ambulant carrier, virus being present in further specimens of stool up to the fourth week. This was as late in the disease as positive results were obtained. The authors record a higher incidence of virus in stools in non-paralytic cases (4 out of 15) than in paralytic cases (4 out of 38). Stools from 20 patients suffering from other acute diseases all gave negative results, as did nine from poliomyelitis contacts. There was a certian amount of difficulty with laboratory animals, chiefly in securing healthy monkeys. The authors used Schweder's palpebral tuberculin test and thus avoided a certain number of tuberculous animals which might not have survived the test. Positive results were obtained in samples of fæces sent from overseas, and during a very hot summer. The virus was present in large amount in one specimen tested.

HYGIENE.

The Foundry Dust Hazard and its Control.

M. F. TRICE (*The American Journal of Public Health and the Nation's Health*, July, 1940) states that in steel foundries certain operations — chiefly mechanical sand conditioning, casting shake-out and cleaning—are responsible for a high atmospheric dust concentration. Instead of the relatively safe concentration of 12,000,000 particles per cubic foot, 40,000,000 to 100,000,000 were found. Of all foundry workers, 1·5% have silicosis. Advanced cases are rare and exposure for a period of thirty years is needed to produce the lesion. Pulmonary fibrosis was noted in 8·2% long before silicosis was present. The incidence of respiratory disease is more than twice the average for industry. More deaths occurred from pneumonia than from any other cause. The author states that the hazards are not confined to silicosis and complicated tuberculosis; for example, a large number of cases of impacted wax in the ear are associated with the dust. Control need not be expensive. Good housekeeping and thorough cleanliness are especially valuable. The monitor type of roof improves ventilation; but exhaust vents are invaluable for the direct removal of dust. Isolation and enclosure of machinery, the use of respirators and positive pressure air helmets, the substitution of steel shot for sand in abrasive cleaning, and regular repairs are all essential. To this must be added examination of the worker before and at periods during employment. Not only improvement in the health of the workman, but also a reduction of about 10% in costs is claimed. Insurance companies allot a premium credit up to 35% for compliance with all these standards.

Immune Reactions of Carriers and Non-Carriers of Type-Specific Pneumococci.

MAXWELL FINLAND, JOHN W. BROWN AND MILDRED W. BARNES (*The American Journal of Hygiene*, September, 1940) have made a bacteriological and immunological study of hospital contacts. They selected a large hospital admitting 500 to 800 patients suffering from pneumococcic pneumonia annually, or about 75 per ward. The subjects were 126 persons — resident medical officers, nurses *et cetera*—who attended the sick and were tested at intervals of one to three months; throat swabs, sputum and blood were used for the tests. Mouse inoculation and agglutination tests were carried out. When one culture only was made, owing to the subject's transfer elsewhere, 36% yielded pneumococci; 56% of all the 126 subjects were found to harbour pneumococci. As a rule, when a positive result was obtained on the first incubation the condition persisted; when positive results were obtained at later tests the condition tended to be temporary. When respiratory infections occurred among these subjects, about one-half showed pneumococci. Types I, VII, VIII, XIII, XVII and XXI occurred more frequently than is usual; Types III, VI and XIII were predominant. In almost every instance characteristic cultures and agglutination features were noted. The Neufeld technique gave the best results. Carriers frequently developed homologous type-specific antibodies. The source of the organism may have been the patient in a few instances; but in many the carriers apparently acquired the pneumococci from their associates. Four of the subjects developed atypical pneumonia; but the organisms were not causally related to the attacks, nor were significant antibodies found.

Prevention and Control of Scarlet Fever.

E. S. BOLTON (*Canadian Public Health Journal*, April, 1940) recommends active immunization against scarlet fever. Scarlet fever toxin was injected every week into school children. After five doses 31% were found to react to the Dick test. A sixth dose of 10,000 skin test doses reduced this to 11%. No serious reaction beyond temporary fever and headache occurred in 12,000 doses. The same needle but two syringes, one for scarlet fever toxoid and the other for diphtheria toxoid, was used. The giving of the two together saves much time, and the three doses of diphtheria toxoid may be readily spaced. In 1939, 3,002 children were at school in Brandon; 50% received scarlet fever toxin. Altogether 69 developed scarlet fever; 12 were in the protected and 57 were in the unprotected group. Of the unprotected, 30% are taken as "Dick-negative", so that the rate in the protected "Dick-negatives" is 6 per 1,000, as against 54 per 1,000 in the unprotected, or 96 fewer cases than the 108 to be expected. The cases among the immunized were very mild and free from serious complications.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XVIII.

THE SEARCH FOR HIDDEN MOTIVES; PSYCHOLOGICAL MECHANISMS; PSYCHOANALYSIS ON THE CONSCIOUS LEVEL.

OWING to the plasticity of the human mind, the motivation of human behaviour, even in childhood, is always to be regarded as far from simple. There is from earliest days a constant interplay between the individual, with his instinctive endowment, and an environment which has to be manipulated in such a way that some satisfaction of instinctive drives is obtained.

There is considerable divergence of opinion as to the nature of our instinctive drives; but there is a firm biological foundation for assuming that the main drive comes from the instincts of self-preservation and sex. These instincts are, of course, far less fixed and automatic than instincts in lower animals, and in course of development the patterns of behaviour which give expression to these drives and lead to the individual's satisfaction, will develop according to experiences encountered and the temperamental characteristics. In addition, there is a process of maturation in the instinctive development so that what would be regarded as a satisfactory adaptation in childhood becomes unsatisfactory in later years.

For example, the satisfaction that the infant obtains by the stimulation of his own body, which, following Freud, we may perhaps regard as sexual, should be left behind to a great extent with the development of normal heterosexual activities. Similarly, the self-preservative instinct develops patterns of behaviour ranging from the infantile sucking at the breast to the manifold activities associated with earning a living and obtaining bodily and mental security and comfort.

There may, however, be hindrances in this development so that some infantile characteristics are shown by behaviour in adult life—for example, unsatisfactory sexual habits or undue dependence on the parents. In these cases the individual is regarded as having a fixation at some earlier stage, and it is found that when difficulties are encountered in later life there is a tendency for the individual to return to this level—there is a regression. Throughout development some satisfaction of our instinctive drives is sought and conflict is inevitable. This conflict may be at different levels. There may be conflict between instinctive tendencies which cannot be satisfied at the same time, or between the instinctive drives and the environment. From these conflicts comes the urge to intellectual and emotional development with the building up of sentiments regarding ourselves and our environment.

Conflict may also arise between the standards we have acquired as to what is right and proper and satisfaction of our instinctive tendencies, and this is a potent source of trouble and deep-seated anxiety.

This estimate of ourselves may be conscious, as an ideal of behaviour to be lived up to; but this is probably to be regarded as merely a superficial aspect of the prohibitions and sanctions of behaviour which have been built in from contacts with parents and others in the early years of development. This larger aspect (super ego) is largely unconscious and seems to be responsible for a most important mental mechanism—repression. Repression has passed into popular usage, but the reality of the process by which the forgetting of facts of experience can be accomplished with no trace in conscious life is not realized by many.

The forgetting of incidents may be due to their triviality—for example, what we had for breakfast a week ago—but it may also be due to the fact that their remembrance would be unpleasant. This at first sight seems absurd, for we can all recall unpleasant happenings; in fact, unpleasant memories may be amongst the most persistent in our minds. Nevertheless the reality of this second kind of forgetting must be appreciated if we are to advance to an understanding of the processes at work in the development of the neuroses and their motivation. This motivation is often hidden from the patients and also from others.

The recognition of motives hidden from others as a conscious act is to be found everywhere from schools to courts of justice, and in literature from detective fiction to the modern psychological novel. Our search for these motives depends on our judgement of facts of behaviour as determined by our knowledge of probabilities from past experience and implies doubt as to the truthfulness of the person concerned. This attitude is present in the minds of many practitioners who think of the apparently useless arm of the hysteric as being a deliberate pretence to attract sympathy and attention, to gain compensation or for some more or less well-hidden motive.

There is, however, no doubt in the minds of most psychiatrists that motives may also be partially or completely hidden from the consciousness of the person concerned, that is, repressed.

It is axiomatic in the Freudian doctrine that no one can know of his repressions excepting through analysis by one who has in his turn been analysed, that is, by someone in "apostolic succession" to Freud himself. The truth of this is uncertain. It is probably impossible for anyone to discover all his repressions even with the assistance of a prolonged analysis, and it is open to most of us to discover by self-examination that there are some other more selfish motives behind acts which at first glance may have seemed wholly altruistic. It is also possible to find some hidden motive or repression at the back of irrational displays of temper, slips of the tongue, lapses of memory and so forth. The imputation of motives such as self-interest, cowardice and so forth in the explanation of the behaviour of others is an even easier task, though it is not always that we are ready to extend the same tolerance to others by assuming that the motives are unconscious. When others ascribe our behaviour to such unworthy motives we are apt to respond with a marked emotional display, denial heaped on denial, counter-accusations and generally speaking rather irrational behaviour, that is, resistance and the development of fresh repressions. Before endeavouring to investigate the hidden motivation of neuroses it is therefore desirable that we should have some realization of this mechanism and of our own weaknesses and that we should endeavour to avoid such resistances in the early stages of investigation by an absence of moralizing, criticism or even advice.

If the patient's super ego has been largely moulded as a result of unfortunate experiences with a harsh, critical, dominating father, it will be seen that the doctor who resembles the patient's mental image of this parent will have great difficulty in making even an initial approach to the patient's real difficulties. This mechanism of repression is one way in which an attempt is made to solve conflicts which cannot be solved on the conscious level.

Another mechanism to be seen in hysterical patients is that of dissociation whereby some portion of mental experience is drastically severed from the rest. This is to be seen in cases of hysterical amnesia in which, following a period of anxiety and conflict, there occurs a sudden break in continuity of thought; some days or weeks later the daily Press reports strange loss of memory by a man of blameless life, who suddenly comes to himself or is discovered working at a new occupation, with no memory of intervening events. Investigation may show that the previous life was not so free from blame and that he had absconded with trust moneys, and the jury's faith, if any, in unconscious motivation may receive a deadly blow. In other cases, however, there is little objective to criticize, and it is only after further investigation that one finds a hidden motive in some deep-seated anxiety and mental conflict which has apparently been responsible for the loss of continuity of memory.

Similarly, the hysterical paraplegias, monoplegias, aphonias, fits, and so forth, may be traced to some preceding anxiety which has been activated by some perhaps quite trivial incident or injury.

In the development of these symptoms we find at work the mechanism of suggestion by which beliefs are firmly implanted without any logical reason for their existence. Examples of this mechanism may be found at all ages; we see it in the young child whose pain is eased by the mother's kiss and assurance that "it's better now", and in the mass propaganda being indulged in at the present time, when anxiety and fear make soil ready for the acceptance of quite illogical beliefs, that is, wishful thinking. In the neurotic this mechanism is very prominent and may be used either as a curative or a harmful agent.

There is a universal tendency to make use of symbols for the expression of ideas which may be extremely complex. Language consists of symbols, the national flag symbolizes a wealth of feeling, and a pound note is a symbol of so much wealth. The same mechanism is freely used in the development of symptoms in hysteria, and a paralysed limb may represent a symbolic solution of mental conflict or indeed of many associated conflicts, for such symptoms may be the "condensation" of many difficulties. An attempt may be made to settle conflicts by the mechanism of "rationalization", in which the resources of our intellect are called upon to justify an action or belief due to unconscious motivation. In other cases some easing of the

conflict is sought in the "projection" of one's unconscious desires on to others.

It will be gathered that there are many difficulties in the way of disentangling the web of causation and arriving at the hidden motives in the background, and to the Freudian analyst the only way of overcoming the resistances and repressions is by the use of "free association". This theory involves the relaxation of the patient and the narration of everything that occurs to him, followed by an interpretation of this by the patient or perhaps by the analyst. This process may extend over years and requires specialist training and experience. It will be found, however, that if the history of the patient and the development of the disability have been taken with a lack of criticism and the patient's confidence has been gained by allowing him freedom to tell his own tale, there may be a stirring up of memories that had been forgotten. Our reward for patience may be a visit from a rather awkward and embarrassed patient who, after considerable hesitation, will say that something occurred to him that he had not thought of for years, and with encouragement a further chapter in the patient's history may be recovered. Perhaps an attempt may be made to evade this by statements that these memories are too trivial to mention; but these valuations are not to be taken at the patient's estimate.

The opening up of such fresh chapters will often lead to considerable benefit, but it is unwise to be too optimistic with apparent successes too easily obtained, for it may be found that fresh repressions will take place and further difficulties arise. With patience and sympathy, however, some progress will be made.

In these partial analyses there is a risk that the patient may develop a "transference neurosis", that is, become emotionally dependent upon the physician to a great extent, but with the maintenance of a policy of "benevolent neutrality" the risk of a complication of this nature to a disabling extent is not great. Such transference neuroses are generally due to an "identification", often on very slender grounds, with someone belonging to the patient's past experience. The patient's statements may give rise to some inference that there are some "blind spots", some rationalizations, projections, repressions and so forth still existent. The drawing of such inferences should not as a rule lead to a blunt declaration that such is the case, but rather the patient should be led to draw such inferences himself. At times, however, such interpretations must be given and must be associated with some reassurance, persuasion or suggestion.

JOHN F. WILLIAMS,
Honorary Psychiatrist, Saint Vincent's Hospital, Melbourne; Honorary Psychiatrist, Children's Hospital, Melbourne.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on July 25, 1940, DR. W. VICKERS, the President-Elect, in the chair.

Acute Rheumatism.

DR. LAURENCE HUGHES read a paper entitled "Acute Rheumatism" (see page 423).

DR. R. A. R. GREEN congratulated Dr. Hughes on his presentation of a subject about which it was difficult to say anything new. The ultimate result of the treatment of acute rheumatism in children hinged on the adequate treatment of the child after the acute stage of the disease had passed. Even in the acute stage medical practitioners found it difficult to persuade parents that rest for such a period as six weeks was necessary. The trouble was that the child often looked well when the acute phase was passed and began to behave with the savagery usually displayed by a convalescent child and was difficult to manage. The action of a practitioner in giving way to the parents by shortening the rest period was often followed by a cardiac tragedy.

Dr. Green was glad that Dr. Hughes had referred to treatment in convalescent homes, for he (Dr. Hughes) had taken an active interest in the establishment of the "Smith Family" home and had done more than anyone else in New South Wales to make continuous treatment effective. Treatment in a convalescent home was the correct line of treatment. It was true that acute rheumatism was not so severe in Australia as in some other countries, and it was also true that full recovery was made relatively easy on account of the equable climate. It had always seemed to Dr. Green that, although the large hospitals, such as the Royal Alexandra Hospital for Children, dealt with many children suffering from acute rheumatism, they had never had proper convalescent homes to which these children might be sent; this was a reflection on the people of the State. Dr. Green went on to say that rheumatism clinics were useful because children could be kept in contact with the medical attendant, the home conditions could be watched and some attempt could be made to improve them. The areas from which rheumatic children came were known as being low-lying and subject to overcrowding. It was obvious that the child's condition could be improved if it were removed to a healthier district. Here almoners were useful, because they knew the financial status of the patient and could give advice on suitable districts. There was no doubt that if better living areas could be found for these children, a real advance could be made. In conclusion, Dr. Green protested that the support given by the public to appeals on behalf of children crippled by orthopædic conditions was out of all proportion to that given to children who could best be described as cardiac cripples. He thought that the cardiac cripple should have just as much provision made for him as one suffering from an obvious crippling deformity.

DR. LINDSAY DEY thanked Dr. Hughes for his comprehensive survey of a subject that was engaging the attention of world authorities. He recalled the fact that when he was in Montreal he found a keen interest being taken in the subject and he was surprised as well as gratified to discover that the pædiatricians in Montreal were quite familiar with the report by Dr. Kempson Maddox that had been published in THE MEDICAL JOURNAL OF AUSTRALIA on the incidence of rheumatism in New South Wales. It seemed to him remarkable that though the climatic conditions of Montreal and the overcrowding in some parts of the city appeared more favourable to the incidence of rheumatism, infection was not more extensive than in Sydney. Dr. Dey went on to remark that the clinician had to be careful to exclude other diseases before a diagnosis of acute rheumatism was made. He thought that this was particularly necessary in monarticular arthritis, which might be due to the presence of a septic condition, such as osteomyelitis in the neighbourhood of the epiphysis or in the joint itself. The child could not be kept too long in bed and the six weeks period should not be lessened, no matter what clinical improvement might appear to have taken place or what the sedimentation rate might be. If this was not done, relapse would occur, and Dr. Dey referred to one or two cases to illustrate what he had said. In conclusion, Dr. Dey said that the convalescent home at Collaroy had not been found suitable for rheumatism patients from the Royal Alexandra Hospital for Children; the home at Pennant Hills was much more suitable.

DR. W. L. CALOV said that he was interested in the climatic factors in the ætiology of rheumatism. It was generally believed that the incidence of acute rheumatism was highest in centres that were cold and wet, and he wondered whether Dr. Hughes had any data which would suggest that the incidence was less in New South Wales than in Victoria, and less again in Queensland than in New South Wales. After several years of residence in the tropics he could recollect having seen only one patient in a tropical country who suffered from mitral stenosis, and even in this instance the diagnosis was by no means certain. Great numbers of post-mortem examinations had been made in Rabaul during his residence there, and, so far as he knew, no case of rheumatic endocarditis had been found. Leonard Rogers, writing of his experience in India, had stated that in 4,800 autopsies he had found only one case of rheumatic endocarditis, and in this instance the sufferer was an Anglo-Indian who presumably had not spent all his life in India. Dr. Calov wondered whether a combination of dietetic, racial and climatic factors might not be responsible.

DR. A. J. NEWTON asked whether it was not possible for the medical attendant by over-emphasis on or by making too much mention of the heart, to give rise to a cardiac neurosis. He asked this question because he had recently seen a recruit, twenty years of age, whose erroneous idea that his heart was affected might have arisen in this way.

Dr. Hughes, in reply, thanked Dr. Green for his remarks and said that he was indebted to him for his unfailing support and ready assistance in the organization of the "Smith Family" convalescent home, and also for his cooperation in the work of the rheumatism clinic. He agreed that it was important that children with acute rheumatism should be away from home. It was not the attending practitioner, but the parents, who objected to the child's removal from home. Dr. Hughes was interested in Dr. Dey's remarks

about conditions in Montreal, and agreed that it was right to insist that the child be kept in bed regardless of what certain clinical tests might reveal.

Dr. Hughes could not answer Dr. Calov's question offhand; he did not know of any investigations carried out in Queensland. Dr. Calov would agree that wetness alone was not an exciting factor in rheumatism, since there was a great deal of wetness in the tropics, but the combination of wet and cold was often stressed in this respect. In reply to Dr. Newton, Dr. Hughes said that if the physician was alive to his duty and if treatment was carried out properly, there would be no over-emphasis upon a child's cardiac condition. Such over-emphasis would be more likely to occur at home than in a convalescent institution; in the latter the child was taught to think of other things than his heart.

DR. W. VICKERS, from the chair, thanked Dr. Hughes for his interesting paper and said that he was glad that the term cripple had been used in connexion with cardiac disability. Cardiac cripples, in his opinion, should receive just as much attention and just as much provision from public funds as those with orthopædic conditions. Dr. Hughes had probably done more than anyone else in Sydney to draw attention to the needs of cardiac cripples and, what was more, he had translated his ideas into action.

A MEETING of the Victorian Branch of the British Medical Association was held at the Mooroopna Base Hospital on June 8, 1940, DR. H. C. COLVILLE, the President, in the chair. The afternoon meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital. Part of the report of this meeting appeared in the issue of October 26, 1940.

Hypertrophic Pyloric Stenosis.

DR. R. O. MILLS showed a female infant, aged three months, in good health and weighing ten pounds two ounces. He said that the birth weight of the baby was six pounds three ounces and that at the age of six weeks, when she came under his care, her weight was six pounds eleven ounces. She had then been ill for ten days, with irregular and increasingly severe vomiting, projectile in type only occasionally. Until the vomiting commenced the baby was entirely breast-fed; but during the ten days prior to her admission to hospital she had been given small additional amounts of Nestlé's condensed milk or cow's milk dilutions. In the early days he had attempted to treat the condition along medical lines, using tincture of belladonna while waiting for eumydrine to be available. Some temporary improvement had occurred both with the belladonna and with the eumydrine solution; but it was not maintained, and the baby again lost weight. Peristaltic waves were not constantly visible on inspection of the abdominal wall while feeding was in progress, but were seen at times. The vomiting was not usually projectile, nor was the amount vomited always anything like the amount of a feeding. Constipation was a feature of the case. He was unable to satisfy himself that he could feel a tumour. He had formed the impression that the tumour should be recognized before operation was advised, and he knew that in hypertrophic pyloric stenosis approximately 80% of the patients were males. He had also read that the weight of the infant at operation was of considerable value in prognosis; in one account it had been stated that of babies under six pounds in weight 75% had died, whereas none died in the group weighing nine pounds or more. Dr. Mills had not made an opaque meal investigation; but it had been shown by test feeding that, on account of the mother's worrying, complementary feeding was required at practically all feedings. After preliminary treatment of the ketosis and dehydration by administration of glucose and saline solution, he had operated and had found a definite pyloric tumour lying well up under the liver, the position explaining the difficulty in palpating the tumour through the abdominal wall. He had done the Rammstedt operation without a preliminary sedative and under general anæsthesia with ether. The baby had commenced since to gain in weight satisfactorily and the progress had continued uninterruptedly.

Dr. Mills remarked that the reports of eumydrine treatment which he had read were variable. It seemed to be regarded as important in Scandinavia and of dubious value in England; he had come across nothing definite about local opinions, and asked for a discussion of the relative values of medical and surgical treatment of the condition.

DR. H. BOYD GRAHAM congratulated Dr. Mills on the accuracy of the diagnosis and on the successful outcome of his treatment. It could be stated with confidence that it was usual in Melbourne to treat such babies surgically after suitable preparation for operation, once the diagnosis of hypertrophic pyloric stenosis was made. The main difficulty that arose, however, was that it was not uncommon to be in honest doubt as to the diagnosis and occasionally to encounter examples of pylorospasm simulating hypertrophic stenosis and readily amenable to medical treatment. Dr. Graham said that he had found eumydrine solution extremely useful as a form of atropine which was less toxic than other forms in combating the spasmodic element; but he was not in favour of its use in hypertrophic pyloric stenosis as a substitute for surgical treatment, though it could be used as an adjuvant. The condition was one in which the cooperation of physician and surgeon in the treatment was of special benefit to the patient.

DR. H. C. COLVILLE said that the patient shown by Dr. Mills illustrated the fact that there was a need to be on guard for the occurrence of hypertrophic pyloric stenosis in the female child. He observed that the presence or absence of a palpable tumour often depended on the doctor's luck in feeling the tumour slipping from under the fingers when he pressed towards the vertebral column. Dr. Colville was in agreement with Dr. Boyd Graham that there was no controversy among pædiatricians in Melbourne concerning the advisability of surgical treatment for the established condition; but even the question of the type of anæsthetic to be used at the operation was often raised for discussion. He could recall in his own experience that at one stage "open ether" anæsthesia was generally employed; that stage was followed by the advocacy of local anæsthesia with the patient strapped to a cruciform splint; and that more recently the surgeons had realized that after all "open ether" anæsthesia was really the best. Dr. Colville went on to describe the pathological condition of definite cartilaginous thickening amounting to considerable hypertrophy on the one hand and the factor of pyloric spasm on the other hand. He said that in hypertrophic stenosis both factors were present and that in a few instances the only pathological condition was the spasmodic one. It was a simple proposition to relieve the spasmodic element, and when the spasm was present alone without the hypertrophy the patients would recover without operation. The improvement that was expected to occur with medical treatment was apt to cause postponement of the operation, and the rule that Dr. Colville observed in a doubtful case was to test the effect of medical treatment for a week or two and then to operate if the baby was not making good progress.

DR. ADRIAN SERONG asked Dr. Colville whether in definite hypertrophy of the pylorus spasm was the essential precursor and the cause of the hypertrophy.

Dr. Colville, in reply, said that the hard cartilaginous hypertrophy was different from hypertrophy in unstriped muscle occurring elsewhere in the alimentary canal, and he found it difficult to believe that it could be brought about by a condition of spasm alone, even if the spasm acted for a long period.

DR. PAUL JONES said with reference to the diagnosis that any baby who was losing weight and vomiting persistently in the first few weeks of life should come under investigation. Visible peristalsis during or after a meal was a valuable sign and the radiographic observations might be diagnostic of stenosis. He expressed the opinion that after the dehydration had been overcome the operation should be performed. He had performed a few such operations. The last patient had died a month after the operation, which was disappointing; the mother had not cooperated sufficiently in carrying out the directions for feeding the baby.

DR. ADRIAN SERONG asked Dr. Boyd Graham to express his views on the treatment of hypertrophic stenosis by means of thickened feedings.

Dr. Boyd Graham said that he was not in favour of the method, because it involved the use of considerable amounts of starch-containing food for very young babies, which was a breach of the principle that such foods should not be given to babies under the age of six months. Other more rational feedings could be used; and the method was apt to be an excuse for unduly postponing the operation by means of which a more rapid, satisfactory and favourable result could be obtained.

DR. DAVID THOMAS said that in Singapore Malayan babies were frequently fed on rice and starch or bananas from the age of a few days; but he had noted that hypertrophic pyloric stenosis occurred in spite of that type of feeding among babies receiving it.

DR. STUART THOMSON asked Dr. Paul Jones for his opinion on the advantages of the transverse incision over the more usual paramedian incision.

Dr. Jones said that he regarded the transverse incision as easier and better than the paramedian incision, because on healing it left an extra fibrous band in the *rectus abdominis* attachments without destruction of muscle fibres. The transverse incision made it easy to deliver the tumour without small bowel coming into the wound; it could be

sewn up readily without interference from the small bowel; and the wound while healing was protected by the underlying liver.

Dr. Mills, in reply, thanked those who had taken part in the discussion; they had discussed the matters on which he had wanted opinions.

Papillary Carcinoma of Thyreoid Origin.

DR. A. L. BENNETT, on behalf of DR. F. W. GRUTZNER, read notes prepared by Dr. Grutzner of the case of a widow, aged eighty-seven years, who had been admitted to the hospital on March 18, 1940, on account of poor general condition with bronchitis and heart failure. She had always been a healthy woman; but approximately three years before her admission to hospital lumps had appeared first on the left side of the neck and later on the other side too. The lumps had caused very little inconvenience; they were not painful or tender; they had varied somewhat in size; and for a few months her voice had been husky. At examination after her admission to hospital it was noted that a massive glandular swelling occupied the left anterior triangle of the neck; two or three smaller glands were found on the right side. The glands were firm, but not hard, more or less discrete, not adherent to the skin and comparatively free from attachment to the deeper structures. In other respects the results of the examination revealed no abnormality apart from the usual signs of senility and mild heart failure. Dr. Grutzner commented that the glandular mass was suggestive of secondary malignant disease; but he had been unable to locate any primary growth. During the patient's stay in hospital there had been no obvious increase in the size of the mass, and indeed he thought that the smaller glands on the right side had diminished somewhat in size. With care and nursing the patient's condition had improved remarkably, and she had even been able to walk about. At the time of the meeting, however, she was suffering from an acute respiratory infection, which was causing considerable anxiety.

Certain clinical investigations had been undertaken. The temperature and pulse rate had been normal except for disturbances due to intercurrent infection. The urine was normal. A blood examination revealed no abnormal features, though the number of leucocytes was low; and the serum had not yielded the Wassermann reaction. One of the smaller glands from the right side of the neck had been removed and sent to Professor MacCallum at the University of Melbourne. Professor MacCallum had reported that examination of sections of the glands revealed considerable hæmorrhage in what appeared to be the tissue between the fairly obvious rounded masses of growth. Where one of the glands had burst the protruding material had a papillary appearance. Microscopic examination of sections revealed that the structure was that of a papillary carcinomatous growth. There was much hæmorrhage throughout the tumour. The papillary structure was very complex and the epithelium covering the papillæ was cuboidal to high columnar in character, and the resemblance of the epithelium to that of thyreoid vesicles was very close. In addition to the extravasated serum there were also areas of accumulation of material with the staining characters of colloid. Throughout the tumour there were, in addition, islands of lymphoid tissue, which suggested in their appearance the type of development frequently seen in abnormal thyreoids. In addition to the papillary arrangement there were also solid areas in which the arrangement suggested vesicle formation. There were abundant mitoses indicating active growth. The condition therefore was one of secondary papillary carcinomatous growth of thyreoid origin.

Dr. Grutzner drew attention to a statement which appeared in the section on diseases of the thyreoid gland in "The British Encyclopædia of Medical Practice", Volume V, and pointed out that those responsible for the section were Professor Fraser and Sir Thomas Dunhill. The quotation was as follows:

> Carcinoma of the thyroid may be of three types: (i) malignant adenoma; (ii) papilliferous adeno-carcinoma; (iii) *carcinoma simplex*. Owing to the capacity of thyroid epithelium to retain its inherited characteristics even when undergoing malignant change the first two of these types tend to overlap . . . in papilliferous adeno-carcinoma a papilliferous growth filling the cystic spaces is the predominating feature and can often be seen with the naked eye. Glands in the neck are involved early. They remain discrete, and dissemination may be restricted to them for a long time. Deposits in glands have the same papilliferous character.

Dr. Grutzner commented that it appeared that it was to the second group that the tumour under discussion belonged. With regard to treatment, it was stated by these authors that the chief hope of controlling the tumours lay in X-ray therapy. A further quotation from the same source was as follows:

> In the meantime it is important to realise that malignant adenoma—which constitutes the vast majority of carcinomas of the thyroid—and papilliferous adeno-carcinoma can be controlled for many years if discovered reasonably early. Two developments have contributed to this better prognosis. One is the recognition of the slower evolution and consequently the longer history of two of the types of malignant tumours of the thyroid, the malignant adenoma and papilliferous adeno-carcinoma; the other is the radio-sensitivity of these types and the advances in the technical skill of radiologists. It may be that further advances in X-ray therapy will supplant surgery altogether, but at the present time surgery is required . . . There is a stage beyond which all treatment is ineffective; but even in some cases which have appeared hopeless life has been prolonged for periods ranging from eighteen months to four years. One patient is alive twenty-four years after the malignant adenoma was removed, although dissemination is now present in the lungs; others are alive and apparently well fourteen years after the first operation.

In his statement Dr. Grutzner then referred to the fact that the patient had not been treated specifically for the condition; her great age and frailty rendered it questionable whether any such treatment would be wise, and there were many practical difficulties in the way. In conclusion he took the opportunity to acknowledge with gratitude the help afforded him by Dr. Stuart Thomson, resident medical officer, in the investigation of the case.

DR. W. M. LEMMON commented on the pathological grouping; malignant adenoma, papilliferous adenocarcinoma, and *carcinoma simplex* were all varieties of the same condition and stages of differentiation of glandular carcinoma with varying malignant trends.

Dr. Colville remarked that the possibilities of successful treatment were obviously not very great. Before closing the meeting he expressed appreciation on behalf of the visitors of the efforts that had been made by the members of the medical staff of the hospital and on the excellent selection and presentation of the cases.

A MEETING of the Surgical Section of the South Australian Branch of the British Medical Association was held on July 10, 1940, at the Royal Adelaide Hospital, Adelaide.

Gastroscopy.

DR. IAN HAMILTON spoke on gastroscopy. He said that gastroscopy was not a new science. Mikulicz, in 1881, knew that diagnostic gastroscopic examinations could be carried out, and he also foresaw that to examine the interior of the stomach a gastroscope would have to be constructed with a distal lamp to illuminate the cavity, and a lens system to bring the light rays up to the eye of the observer. Many types of gastroscope had been constructed; some were simple open tubes, some were rigid, with an obturator for introduction and a telescope for viewing like a cystoscope. Others were flexible and had an appliance for straightening them after introduction; but although a great amount of work had been done on the instruments by 1921, practically no one used gastroscopy at that time as a diagnostic method. The work was taken up by Schindler in Münich. He used straight rigid tubes at first and published the results of a large series of gastroscopic examinations. However, he was never satisfied with these instruments on account of the grave danger of rupturing the lower end of the œsophagus with them. With the help of the great genius of the optical scientist Wolf, by 1932 Schindler had produced the Wolf-Schindler flexible gastroscope. That extraordinary instrument was partly flexible and partly rigid. The upper part, extending nearly down to the cardia, was rigid. The distal portion from about three centimetres above the cardia was flexible and elastic. It carried a rubber finger at its tip, a strong electric light, and just proximal to this the objective. The objective picked up the light at an angle of 90°, with a field varying from 50° to 85° in different models, and carried it up through the flexible and rigid parts to the eyepiece by means of 28 lenses. A beautiful clear image could be seen provided that the instrument was not bent beyond 34°; at that degree of bending the image became oval and it was lost at 35°. The instrument was extremely delicate and expensive.

Dr. Hamilton went on to say that the most difficult parts of the technique were those concerning the passage of the gastroscope and then of orientation when the interior of the stomach was to be viewed. The technique had to be carefully

learnt, and many normal stomachs had to be examined so that one could accustom oneself to the normal variations seen. The pylorus should be seen in eight out of ten examinations; but the beginner could never hope to accomplish this. Very slight variations in the position of the patient made a great difference in the amount that could be seen. An experienced nurse was necessary to hold the patient's head. With careful preparation and anæsthetization most patients could be successfully, painlessly and satisfactorily examined; but Dr. Hamilton had failed in two cases. In one it was impossible to give the anæsthetic because the patient, a nervous female, would not allow the attendants to paint her gums and tongue; in the other case the anæsthetic was given, but through nervousness there was such a pronounced spasm of the *musculus constrictor pharyngis inferior*, a strong muscle quite capable of preventing the passage of the instrument if thrown into spasm, that Dr. Hamilton was unable to pass the instrument into the œsophagus. Dr. Hamilton had performed gastroscopic examinations 34 times, and some three or four patients had had several examinations; beyond slight soreness in the throat there had been no complaints. Dr. Hamilton was informed afterwards that one man thought he should have been kept in hospital after the examination instead of being sent out straight away. No accident had occurred, and in those cases in which a subsequent check by operation was made, the diagnosis usually proved correct.

Dr. Hamilton then said that he was often asked what area of the stomach could not be seen; in that connexion, illustrations from the works of Moersch and Harold Edwards were valuable. In point of fact, most of the mucosa could be viewed. Gastroscopy was a supplementary diagnostic method. It was not to be compared in the full sense with radiography, because it did not compete with it. Radiography gave pictures of the whole alimentary tract; gastroscopy revealed only the stomach; but in the stomach it appeared to Dr. Hamilton to reign supreme in regard to diagnosis.

Dr. Hamilton then referred to Schindler's statements concerning gastroscopy. These were the following: (i) The foundation of all his findings was his knowledge of the normal stomach. (ii) An ulcer of the stomach was usually well seen; an ulcer of the duodenum could not be seen. (iii) Carcinoma of the stomach showed a great variety of forms and could usually be satisfactorily distinguished from simple ulcer. Its operability might be gauged. (iv) Chronic gastritis could be satisfactorily diagnosed only with the gastroscope. In that connexion Gutzeit, quoted by Moersch, had stated that chronic gastritis was twelve times as common as gastric ulcer and three times as common as duodenal ulcer. Chronic gastritis was divided by Schindler into (*a*) superficial gastritis, (*b*) atrophic gastritis, and (*c*) hypertrophic gastritis. (v) The stomach after operation could be viewed in such a manner as to give extremely valuable information.

Dr. Hamilton went on to say that during his travels in America he discussed gastroscopy with many prominent surgeons and some physicians. To see Schindler himself performing the examination was to see an absolute master. Dr. Hamilton had never seen a more dexterous expert than he. In the hands of such a man gastroscopy was, he believed, of enormous value. In the hands of some workers whom he saw it appeared quite valueless; and perhaps because they had not seen Schindler at work they could not improve their methods. The Mayo Clinic employed a good endoscopist, Moersch, and the physician Eusterman had told Dr. Hamilton that his work was very useful as an aid to diagnosis in certain obscure cases. Moersch did not believe in making a diagnosis unless he could see some positive foundation for it, and he had expressed the opinion that direct visualization of the stomach should be attempted when clinical and radiological evidence was inconclusive or negative, although there was symptomatic evidence of intrinsic gastric disease. A noted surgeon in New York, whose partner was making gastroscopic examinations, seemed very disappointed with the results. Most surgeons had only heard of the method and were afraid of rupture of the œsophagus. However, such was Schindler's fame that all his courses of instruction in gastroscopy were booked up months ahead, and only those who confined their work to internal disease or gastroenterology were accepted by the University of Chicago for such instruction. A gastroscopist from Guy's Hospital whom Dr. Hamilton had met at Schindler's clinic, had informed him that he received so many requests from the physicians of that institution for gastroscopic opinions that he had difficulty in keeping up with them. Dr. Hamilton had formed the opinion that gastroscopy had come to form one of the greatest aids to diagnosis in gastric disease, but that to be effective it had to be properly organized, and the technique as taught by Schindler had to be thoroughly mastered.

Dr. Hamilton then performed gastroscopic examinations on two patients. The first was a woman, aged forty-four years, who had suffered from what she described as "gastric trouble" for two and a half years. The symptoms were pain in the right side of the upper portion of the abdomen and regurgitation of food. Examination after a test meal disclosed achlorhydria, and a cholecystogram revealed no abnormality. The patient had been relieved by the administration of dilute hydrochloric acid. The gastroscopic appearances were those of a normal gastric mucosa.

The second patient was a man, aged fifty-two years, who for eighteen months had had periodical attacks of epigastric pain associated with vomiting. He had lost weight. X-ray examination revealed a questionable filling defect on the lesser curvature of the stomach just above the pylorus. Examination after a test meal revealed a normal acid curve. Gastroscopic examination disclosed an area of infiltration on the lesser curve towards the pylorus. Here the mucosa appeared whiter than normal, with a marked degree of lumpiness and inelasticity. Beside this area a small crater was seen. The appearances were strongly in favour of a diagnosis of carcinomatous infiltration, which supported the radiologist's suggestion.

Partial Gastrectomy for Peptic Ulcer.

Dr. Hamilton also showed four patients who had undergone partial gastrectomy for complicated chronic peptic ulcer. They all expressed themselves as feeling exceedingly well, their only symptoms being a slight sensation of distension after a large meal. All had put on weight and could eat anything they fancied, and they all felt strong and hearty.

The first of the four patients was a man, aged seventy-three years. He had suffered from "wind in the stomach" for five years and had had a hæmatemesis five years earlier. He had had epigastric pain half an hour after meals and had lost a stone in weight. A small mass could be felt in the region of the pylorus, and the results of X-ray examination were indefinite. Gastroscopy revealed a normal stomach, but the pylorus was not visualized. A test for occult blood in the fæces produced a positive result. On September 29, 1939, Dr. Hamilton operated and found a large penetrating ulcer, about one centimetre in diameter, on the postero-medial aspect of the duodenum. The head of the pancreas was thickened, firm and rather nodular; this was apparently due to a chronic pancreatitis where the ulcer had perforated into the pancreas. An anterior isoperistaltic partial gastrectomy of the Pólya-Balfour type and partial duodenectomy were performed. Recovery was uneventful.

The second of the four ulcer patients was a male, aged forty-seven years, who had had indigestion, pain in the right side of the epigastrium, wind, eructations and vomiting intermittently for ten years. He had had severe hæmatemesis. An X-ray examination revealed a large penetrating ulcer with obstruction at the pylorus. Examination after a test meal revealed persistent hyperchlorhydria. A gastroscopic examination failed to reveal the ulcer. Operation was performed on December 8, 1939, and revealed a large ulcer cavity lined with thick fibrous tissue, the size of a billiard ball, right in the substance of the left lobe of the liver, communicating with the stomach by a small hole which just admitted the tip of the little finger, just proximal to and above the pylorus. There were many firm adhesions all around the area. The ulcer was cut away from the liver and an anterior Pólya-Balfour isoperistaltic partial gastrectomy was performed. The patient's convalescence was marred only by a small abscess in the wound, and he was discharged from hospital four weeks after operation.

The next ulcer patient was a male, aged sixty-three years, who had had upper abdominal pain for several years. In December, 1937, an X-ray examination revealed a large penetrating ulcer on the lesser curvature of the stomach. A wedge excision was performed at that time, but the ulcer soon recurred with an accompanying hour-glass contraction of the stomach. On March 31, 1939, another operation was performed; a large ulcer was found on the posterior surface of the stomach near the lesser curve and attached to the pancreas at its base. There were numerous adhesions between the stomach and liver. A partial gastrectomy of the anterior Pólya-Balfour isoperistaltic type was performed. Recovery was entirely uneventful. In the interlude between this patient's two gastric operations he fell from a ladder and fractured his left *os calcis*. The joint was subjected to subastragaloid arthrodesis, with a very satisfactory result.

The final ulcer patient was a male, aged forty-eight years, who gave a history of upper abdominal pain two hours after meals, distension and flatulence, on and off for eighteen months. X-ray examination revealed a large penetrating ulcer just above the *incisura angularis*, strongly suggestive of carcinoma. The patient was operated on on May 1, 1936, when a deep indurated ulcer, half an inch in depth and three-quarters of an inch in diameter, was found on the lesser curve of the stomach; it was densely adherent by a strong fibrous mass to the posterior wall of the lesser sac.

A partial gastrectomy was performed by the anterior Pólya-Balfour isoperistaltic technique, and recovery was uneventful.

In regard to all these patients, Dr. Hamilton drew attention to the important fact that he had chosen the anterior Pólya-Balfour technique for all the partial gastrectomies, except when physical conditions dictated the need for some other procedure, and that the results had been extremely gratifying.

Nævus Treated by Skin Grafting.

Dr. Hamilton also showed a girl, aged sixteen years, who had had a large disfiguring nævus on the nose. This had been completely excised and a whole thickness free skin graft had been used to replace the skin. The whole graft had lived, and although it was only twelve days since the operation the appearance was very satisfactory.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 213, of October 17, 1940.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Appointments.—Surgeon Lieutenant (for Short Service) Francis Harrison Genge is appointed Surgeon Lieutenant on the Permanent List, with seniority in rank of 4th March, 1935, dated 4th November, 1940.

Promotions.—Surgeon Lieutenant-Commander Hill Gillman Wells is promoted to the rank of Surgeon Commander, dated 18th September, 1940.

Emergency List.

Termination of Appointment.—The appointment of Surgeon Lieutenant Gerald Carew Moss is terminated, dated 29th June, 1940.

AUSTRALIAN MILITARY FORCES.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captains.—John Howard Simmonds, 29th August, 1940; Nathaniel Sydney Corrigan, Kenneth John Ward and Ernest Nicholls Kurrle, 30th August, 1940; Robert Allan Spence, 11th September, 1940; and William Elvyn Hood, 19th September, 1940. Lieutenant G. B. Ferguson is transferred from 42nd Battalion and to be Honorary Captain, 19th August, 1940.

EASTERN COMMAND.

Second Military District.

Australian Army Medical Corps.

To be Majors (temporarily)—Captains R. B. Perkins and Captain (provisionally) J. B. McElhone, 18th September, 1940. *To be Captains (provisionally)*—John Kenneth Mackenzie, 23rd August, 1940; John Ziegler Huie, George Alfred Hodgson, Norman Arthur Walker, Austin Arthur Callen, Patteson Omeo Armytage, James Murray Gordon Pirie, Alfred Kenneth Jones, Saywell Clifton Barrett, Sidney Brookes Clipsham, Alan Sidney Brett, Francis Henry McClements Callow, Malcolm Livingston Edwards and John Joseph Gearin, 6th September, 1940; Norman Roy Paterson, Robert John Walsh and Mervyn Norman Tinckham, 9th September, 1940; Theodore Jack Constance, 13th September, 1940; Norman Ernest Davis, 19th September, 1940. Major A. Goldstein is appointed from the Reserve of Officers (A.A.M.C.), 13th September, 1940; Honorary Captain R. C. Geeves is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 21st September, 1940; Honorary Captain A. J. Shappere is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 27th July, 1940 (this cancels the notification respecting this officer which appeared in Executive Minute No. 175/1940, promulgated in *Commonwealth Gazette*, No. 190, of 12th September, 1940).

Australian Army Medical Corps Reserve.

To be Honorary Captains—John Ernest Burtinshaw, 31st July, 1940; Leslie Utz, 6th September, 1940; Esmond Clifton Smith, 11th September, 1940; Keith Klein and Rowland Champion Thomas, 13th September, 1940; David Leonidas Cropp, 16th September, 1940. Honorary Captain F. E. Wall is retired.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

To be Captains (provisionally)—Alexander John Maum Sinclair, 30th August, 1940; Kenneth Fitzpatrick Russell, 14th September, 1940; John Watson, Henry Devenish Steward and Alexander Hugh McGregor, 23rd September, 1940.

Australian Army Medical Corps Reserve.

To be Captain—Alfred John Trinca, 14th September, 1940. *To be Honorary Captains*—James Mitchell Sleeman, Anthony Richard Kevin Kelly, Cyril Hopetoun Dickson, Michael Francis Addison Woodruff, Cecil Ashley Cohen, John Alexander Blitz, Mostyn Levi Powell, Bernard Allen Baker, Victor Edward Sampson, Francis Michael Blackall and Allan Finlay MacInnes, 14th September, 1940; Harley John Enniss, William Ernest King, Charles Edward Richardson, Harold Newton Zimmer and Edmond Clarke, 23rd September, 1940; Winifred Iris Evelyn MacKenzie and Allan Aveling Ferris, 25th September, 1940. The resignation of Honorary Captain R. L. T. Grant of his commission is accepted.

Fourth Military District.

Australian Army Medical Corps.

To be Major (temporarily)—Captain F. E. Welch, 22nd August, 1940. *To be Captain (provisionally)*—George Norman Lorimer, M.C., 18th May, 1940. Honorary Captain R. H. Formby is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 22nd June, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Arthur Douglas Reid and David Wickham Shepherd, 20th June, 1940; Harry Max Wilson and James Henry Vanstone, 26th June, 1940; William Rosslyn Charles Morris, 3rd July, 1940; Rex James Sargent, 16th July, 1940; Brian Herbert Swift, 17th July, 1940; Harold Richard Hugh Noel Oaten, 22nd July, 1940; Hugh McIntyre Birch, 24th July, 1940; Max Ernest Hannaford Schafer, 27th July, 1940; Donald James Robert Sumner and Frank Rees Magarey, 19th August, 1940; Arthur Mervyn Myers, 20th August, 1940; Robert Hugo Elix, 2nd September, 1940; Neill Horace Munday, Leslie Wadmore Linn and Richard Constantine Bassett, 3rd September, 1940; Kenneth Stuart Hetzel, 7th September, 1940; Colin Percival Juttner, 10th September, 1940; Frederick Edward Terrill, 14th September, 1940, and Frederick Lewis Thyer, 20th September, 1940. Honorary Captain M. E. Goode is retired.

Sixth Military District.

Australian Army Medical Corps Reserve.

To be Captain—James Magner, 12th August, 1940.

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps Reserve.

To be Honorary Major—Frank Llewellyn Gill, 29th May, 1940. *To be Honorary Captain*—Alwyn Gerald Davies, 18th September, 1940.—(Ex. Min. No. 198—Approved 15th October, 1940.)

Correspondence.

THE RELEASE OF FIXED "RECORD" SYRINGES.

SIR: A method I have found highly successful is to place the syringe in a bath of ether containing a few drops of olive oil.

I have never tried its use where metal meets metal at the top of the syringe, but I fancy it would meet this case where the refrigerator method would, no doubt, fail.

The ether is the vehicle which carries the fine layer of oil between the opposing parts.

Yours, etc.,

L. J. SPENCE.

17, Bolton Street,
Newcastle,
New South Wales.
October 18, 1940.

NATIONAL FITNESS.

SIR: The reference in your leading article to the national fitness campaign is indeed welcome. In Victoria the movement promises well, but two circumstances require attention.

The Federal Government grant of £1,000 a year is inadequate for the excessive work which is being undertaken in Victoria by the council and the organizer, Dr. Scholes. It has become further obvious that success will only be achieved by the cooperation of the municipal councils, who are responding well.

All sorts of problems arise. Want of open-air spaces, supervised playgrounds, malnutrition, youth hostels in the country *et cetera*. A visit to Brighton is an object lesson in the success of the council's effort and its capable engineer.

But it is obvious that the full support of the medical profession is wanted, and also some more money for the central body. The total expense need not be very great if people and organizations cooperate.

Yours, etc.,

103-105, Collins Street, Melbourne, October 21, 1940. JAMES W. BARRETT.

Obituary.

SARAH VICTORIA McCONNEL.

WE regret to announce the death of Dr. Sarah Victoria McConnel, which occurred on October 16, 1940, at Stanthorpe, Queensland.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Bodley, Phyllis Ellen, M.B., B.S., 1939 (Univ. Durham), 172, Pacific Highway, North Sydney.

Langley, Alan Leslie, L.R.C.P., L.R.C.S. (Edinburgh), L.R.F.P.S. (Glasgow), 1937, All Saints Rectory, Ocean Street, Woollahra.

Sands, Clive Chisholm, M.B., B.S., 1940 (Univ. Sydney), 38, Shirley Road, Roseville.

Windsor-McLean, Leonard Allan, M.B., B.S., 1932 (Univ. Adelaide), D.T.M., 1935 (Univ. Sydney), St. George District Hospital, Kogarah.

QUEENSLAND.

THE undermentioned has been registered, pursuant to the provisions of *The Medical Act*, 1939, of Queensland, as a duly qualified medical practitioner:

Lee, Milton Raymond, M.B., B.S., 1938 (Univ. Sydney), c.o. Dr. S. A. McDonnell, St. George.

The following additional qualifications have been registered:

Cilento, Sir Raphael West, D.T.M. and H., R.C.P. and S. (England and London), 1922, Ipswich Road and Villa Street, Annerley.

Carseldine, Malcolm Wesley, Adelaide Street and New Sandgate Road, Clayfield, N.2. (M.B., B.S., 1929, Univ. Sydney), M.S., 1940 (Univ. Queensland).

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Bodley, Phyllis Ellen, M.B., B.S., 1939 (Univ. Durham), "Woodstock", 172, Pacific Highway, North Sydney.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Deery, Arthur, M.D., 1933 (Univ. Padua), Quambatook, Victoria.

Medical Appointments.

Dr. W. W. Jolly has been appointed a Temporary Honorary Assistant Surgeon at the Royal Adelaide Hospital, Adelaide, South Australia.

Diary for the Month.

Nov. 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 6.—Western Australian Branch, B.M.A.: Council.
Nov. 6.—Victorian Branch, B.M.A.: Branch.
Nov. 7.—South Australian Branch, B.M.A.: Council.
Nov. 8.—Queensland Branch, B.M.A.: Council.
Nov. 12.—Tasmanian Branch, B.M.A.: Branch.
Nov. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 19.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 20.—Western Australian Branch, B.M.A.: Branch.
Nov. 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 22.—Queensland Branch, B.M.A.: Council.
Nov. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 27.—Victorian Branch, B.M.A.: Council.
Nov. 28.—South Australian Branch, B.M.A.: Branch.
Nov. 28.—New South Wales Branch, B.M.A.: Branch.
Nov. 29.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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